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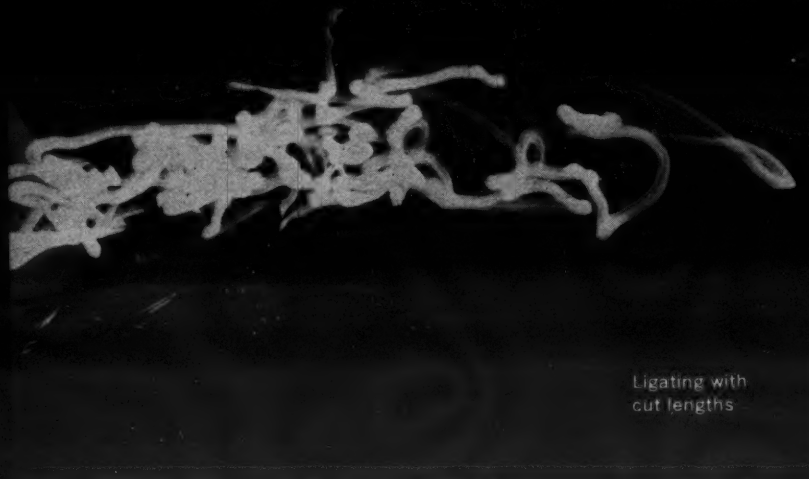

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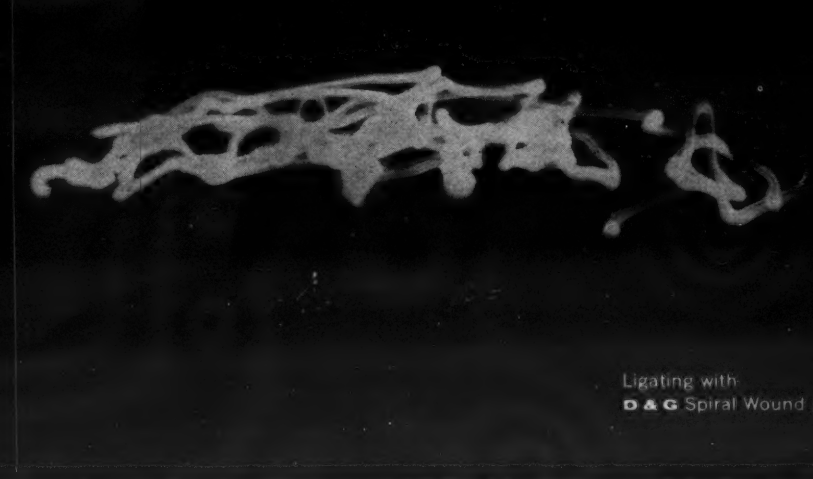

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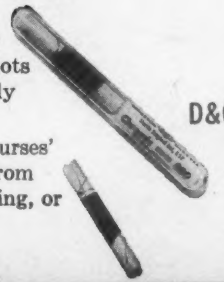

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EXPERIMENTAL VASCULAR GRAFTS. X: STUDIES OF FRESH VENOUS AUTOGRAFTS TREATED WITH HEVEA LATEX OR PLICATION[1]

RALPH K. ZECH, M.D.[2], LLOYD M. NYHUS, M.D.[3], GILBERT G. EADE, M.D.[4], HENRY N. HARKINS, M.D., PH.D., F.A.C.S.[5]

Seattle, Wash.

An increasing awareness that many types of arterial disease are now amenable to surgical correction has given impetus to the field of vascular surgery. A great need thus has arisen for suitable substitutes to replace excised segments of the arterial system. The most desirable replacement for use as an arterial graft would be an autogenous artery; however, the extensive segment of vessel needed usually negates this source. Schmitz, Merendino, Kiriluk, Kanar, and Harkins (1953), Potts, Albert, and Fischer (1953), Holman and Hahn (1953), Hurwitt and Kantrowitz (1952), and Sandblom and Ekstrom (1952) have devised ingenious methods for increasing the caliber of arterial grafts, but the clinical application of these methods is restricted.

Homologous arterial grafts have been widely employed and the results are encouraging, although it is still early for a definitive statement as to their eventual fate. Experimentally at least, inadequate growth and degenerative changes have been frequent accompaniments of arterial homografts, Nyhus, Kanar, Moore, Schmitz, Sauvage, and Harkins (1953), and Kanar, Nyhus, Schmitz, Sauvage, Moore, Zech, and Harkins (1954).

The introduction of prosthetic replacements, Voorhees, Jeretzki, and Blakemore (1952), for arterial segments offers a new and promising source of usable implants. However, related studies are not of a decisive nature at this time.

[1] From the Department of Surgery, University of Washington School of Medicine, Seattle. This work was aided in part by a grant (H-1136) from the Division of Research Grants, National Institutes of Health, Bethesda, Maryland, and by Initiative 171 Research Funds of the State of Washington.

[2] Formerly National Heart Trainee, National Institutes of Health, and Instructor in Surgery.

[3] Instructor in Surgery and Guggenheim Fellow in Surgery, 1955–1956.

[4] Postdoctorate Research Fellow, Public Health Service, and Assistant in Surgery.

[5] Professor and Executive Officer, Department of Surgery.

There have been numerous experimental studies related to autogenous tissue replacement of arterial defects. Autogenous tissue conduits have been created from pericardium, Sako (1951), and Zech, Nyhus, Griffith, and Harkins (1955), fascia, Cousar and Lam (1952), and vein, Nyhus, Kanar, Moore, Schmitz, Sauvage, Zech, and Harkins (1955). These autogenous tissues tend to maintain an adequate rate of growth; however, their use has been complicated by a definite tendency to dilate. Experimental methods have been utilized in an endeavor to prevent this dilatation of autogenous tissue. The results of using fascial supports, Sako (1951), Zech, Nyhus, Griffith, and Harkins (1955), viable musculofascial support, Nyhus, Moore, Kanar, Zech, Griffith, and Harkins (1955), and perivascular fibroblastic agents, Zech, Nyhus, Kanar, Schmitz, Sauvage, Moore, Merendino, and Harkins (1954), to prevent dilatation of these autogenous tissues have been equivocal.

We believe that studies related to autogenous tissue grafts should be extended. We have accordingly investigated two experimental methods in an attempt to prevent dilatation of autogenous venous grafts of the thoracic aorta of growing pigs. Woodhall and Golden (1953) in a series of experiments found that when hevea latex was applied to the arterial walls of rabbits, there was a uniform and thick fibrosis of the adventitia without intrinsic alteration of the vessel's function. With these latter experiments in mind, we hoped to prevent venous autograft dilatation by uniform thick adventitial fibrosis produced with application of hevea latex.

In the experiments related to fresh unsupported venous autografts which were reported from our laboratory in 1955, Nyhus, Kanar, Moore, Schmitz, Sauvage, Zech, and Harkins, it was noted that the three venous autografts which were of near isodimensional proportions and without aneurysmal dilatation at the time of graft harvest were about 25 per cent smaller than the host aorta at the time of implantation. On the basis of these observations, it was deemed advisable to decrease purposely the external diameter of a few venous autografts at the time of graft implantation in order to investigate the possible effect of "bilateral negative disproportion" of the graft upon this incidence of subsequent graft dilatation.

METHOD

Thirteen weanling pigs with an average weight of 12.8 Kg. were subjected to a retroperitoneal resection of the inferior vena cava. The excised venous segment was implanted into a surgically created defect in the thoracic aortas of these same weanling pigs. Careful measurements were recorded. All procedures were performed in one stage under aseptic conditions.

After the fresh venous autografts had been implanted, 7 were carefully excluded from the adjacent tissues and bathed in a layer of hevea latex. Care was taken to have the latex contact only the exterior surfaces of the graft. All latex covered grafts were well pleuralized after implantation.

The remaining 6 grafts were plicated by the use of interrupted Lembert sutures of no. 00000 oiled arterial silk utilizing the same technics as described by

Schmitz, Sauvage, Kanar, and Harkins (1953). The plication sutures were placed about 0.2 cm. apart. When the plication sutures were tied, it caused an infolding of the graft wall (figure 1). The external diameter of the plicated venous autografts averaged about 25 per cent less than the adjacent host aorta. Most sutures excluded the intima; however, light digital pressure easily controlled the few

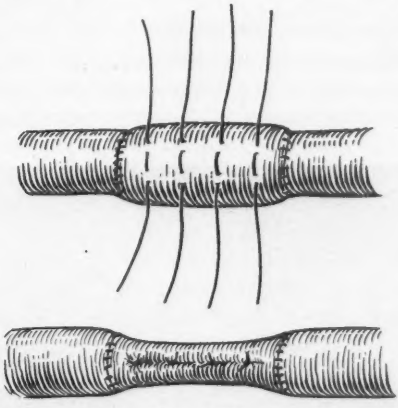

FIG. 1. Upper: Schematic presentation of the procedure of plication. Note that the graft has a biterminally positive disproportion. Lower: Plication completed. The graft is now biterminally negative. The sutures originally placed caused an infolding of the graft wall and reduced the caliber of the graft. The intima was excluded in most instances.

TABLE I

Summary of the dimensional changes in fresh venous autografts treated, at the time of implantation into the thoracic aorta of growing pigs, with hevea latex or plication

Pig No.	Procedure	Per Cent Weight Gain	Per Cent Linear Growth			Per Cent Diameter Growth	
			Animal	Aorta	Graft	Aorta	Venous Graft
201	Latex	1050	125	141	141	110	425
207	Latex	750	105	155	30	125	40
212	Latex	785	84	110	21	90	270
226	Latex	750	71	—	57	90	288
238	Latex	990	83	105	100	110	140
214	Plicated	600	64	67	76	86	175
215	Plicated	1020	110	120	44	125	200
216	Plicated	815	76	80	95	83	285
217	Plicated	700	52	—	61	170	185-355
224	Plicated	1150	Torn in removal but qualitative observations made				
231	Plicated	845	100	115	130	130	450
208	Latex	—*	43	40	48	30	0

* This animal died 4 months after surgery of pneumonia. There was no scale available to obtain its weight.

bleeding areas which were encountered when the plication suture inadvertently pierced the intima.

Following recovery from the surgery the animals were transferred to a boarding farm where they were allowed to mature to market size. The grafts were harvested at the time of slaughter. All necessary measurements, photographs, and microscopic sections were then completed.

RESULTS

There were no operative deaths. Eleven animals reached maturity; 2 animals with latex treated venous grafts died, 1 at 6 weeks of occlusion of the graft by a small mural thrombus superimposed upon a greatly thickened fibrotic graft wall.

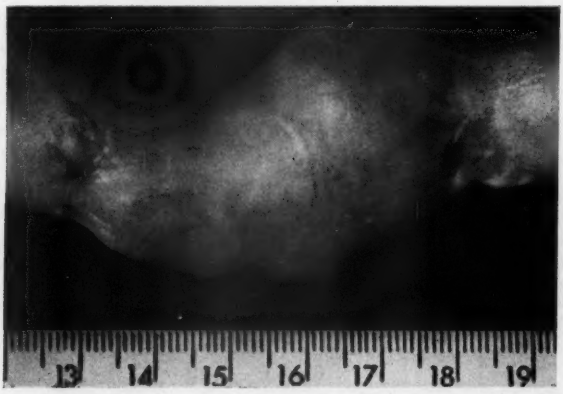

FIG. 2a

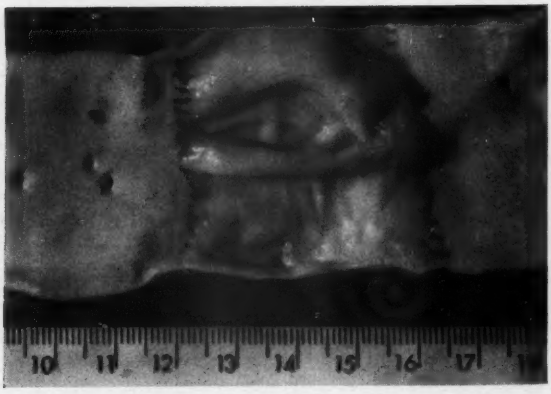

FIG. 2b

FIG. 2. Photographs taken of Pig Graft 226, a fresh venous autograft of the thoracic aorta bathed in liquid latex. a. Inflated graft (120 mm. Hg with air). The graft is diffusely dilated and has daughter aneurysms. Note how the anastomotic suture has straightened with growth. b. Open view: The intima is smooth and the sutures are well endothelialized. Note that the graft wall is nearly as thick as the adjacent aorta. A large aneurysm is visible.

The other animal died of lobar pneumonia unrelated to the isodimensional and patent graft. Microscopic sections were taken of both these grafts.

1. *Latex Supported Venous Autografts of the Thoracic Aorta of Growing Pigs:* Five animals reached maturity, all were healthy, and free of infection and neoplasm. One of these grafts (Pig 207) was occluded by a web of organized thrombus. Animal growth occurred at a normal rate in these animals; they increased their weight an average of 865 per cent.

Table I presents comparative dimensional growth changes for both series of animals. It should be noted that most latex treated grafts failed to maintain a rate of linear growth compatible with either the animal as a whole or with the adjacent aorta. There was, however, a consistent and prominent increase in the diameters of all grafts except the one which was thrombosed (Pig 207), in which there was a general retardation of growth. These diameter changes often manifested themselves as diffuse or fusiform expansions, and daughter aneurysms were common (figure 2).

The walls of the latex treated grafts were thick and fibrotic; the localized dilatations were, however, thinner than the adjacent aorta. There was some thinning of the graft at the suture lines, the pliability of the implants was reduced, and there were no signs of infection.

Microscopic sections revealed symmetric thickening of the graft adventitia by a dense fibrous tissue sheath having interspersed areas of collagen deposition.

FIG. 3. Photomicrograph: Latex supported venous autograft of thoracic aorta of Pig 205. This graft was implanted for 6 weeks. H & E stain × 25 (Intimal surface up): There is thrombus formation with an intimal covering. Note the inflammatory cellular reaction which is composed principally of mononuclear cells. There are several giant cells and interspersed meshes of eosinophils. There is definite thickening of the adventitia, the result of a rather symmetric deposition of fibrous tissue. A Weigert's stained section not shown demonstrated preservation of the elastic tissue fibers.

FIG. 4. Photomicrograph: Latex supported venous autograft of the thoracic aorta of Pig 208, 4 months postimplantation. H & E stain × 25 (Intimal surface up): There is definite thickening of the graft wall. The intima appears normal. The graft wall is composed of elastic tissue fibers enmeshed in a rather dense fibrous tissue having interspersed areas of collagen deposition. The inflammatory reaction is subsiding although several giant cells remain and mononuclear invasion is prominent. There is early vascularization of the adventitia and eosinophils are still present.

Six-week sections (figure 3) showed in addition a moderate mononuclear cell infiltration with occasional giant cells and eosinophils scattered throughout this reactive tissue. The adventitia revealed a lessening cellular reaction as the graft matured. The fibrous tissue became more compact and there was evidence of adventitial vascularization (figure 4).

2. *Plicated Venous Autografts of the Thoracic Aorta of Growing Pigs:* All 6 animals of this series reached maturity; all were healthy and free of infection. The pigs matured at a satisfactory rate, averaging a 750 per cent weight gain. All the grafts were patent. Linear growth of the graft approximated that of the adjacent aorta, whereas those grafts bathed in latex had a suppressed growth when compared to the growth of the adjacent aorta.

There were definite dilatations present in all grafts (figure 5), but these changes were neither so prominent nor so severe as were those noted with the latex series.

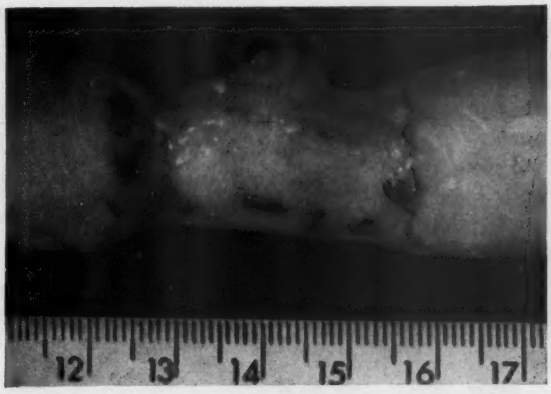

FIG. 5a

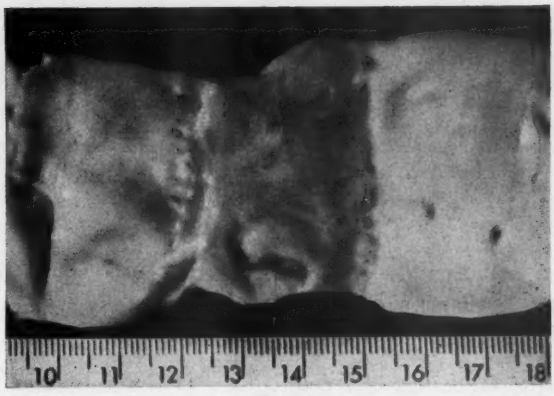

FIG. 5b

FIG. 5. Photograph taken of the mature graft implanted into the thoracic aorta of Pig 215. This graft was plicated. a. Inflated view (120 mm. Hg with air). There is prominent aneurysm formation at the midgraft area; the remainder is almost isodimensional. The plicated sutures are visible along the inferior margin. The Carrel anastomotic suture has straightened with growth. b. Open view: All sutures are well endothelialized; the intima is quite smooth. The graft is thin near the lower anastomosis (right). The aneurysm is visible in the middle third of the graft.

The graft walls were thinner than the adjacent aortic walls and the grafts were generally dilated and presented daughter aneurysms which were usually located on the anterior surface of the graft. The adventitial reaction was minimal. The intima was smooth and glistening; the suture lines were well endothelialized, and the furrow formed by the plication was obliterated. There were no thrombi.

Microscopically, plicated grafts were similar to other venous autografts of the thoracic aorta of growing pigs, Sauvage and Harkins (1954). The grafts were in essence endothelialized connective tissue tubes. Elastic tissue fibers were present, while smooth muscle cells were infrequently found (figure 6). In all the auto-

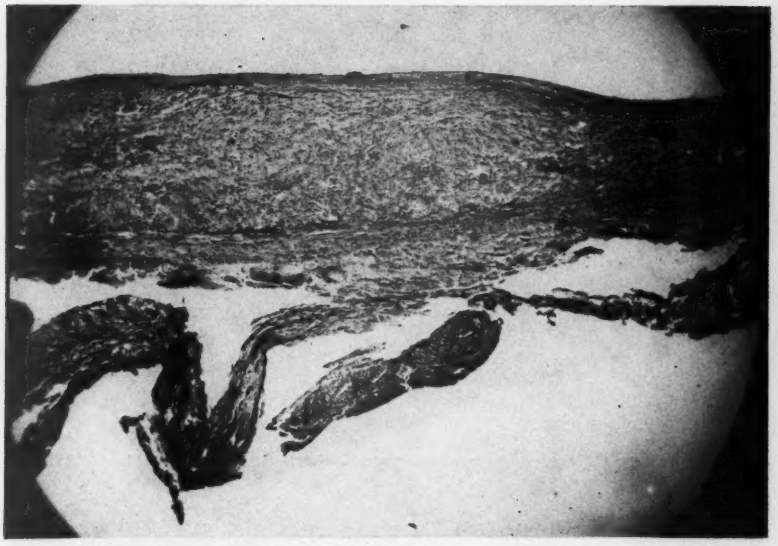

FIG. 6. Photomicrograph: Latex supported venous autograft of the thoracic aorta of Pig 212, 8 months after implantation. a. H & E stain × 20 (intimal surface up): The intima is intact. The fibrous tissue deposited earlier has contracted and there are prominent areas of collagen deposition. The inflammatory cell invasion is greatly reduced and is composed almost entirely of mononuclear cells. The graft wall has thinned as compared with earlier stages, but it is now composed of a dense fibrous tissue. Adventitial vascularization is well established. Elastic tissue fibers remain.

grafts observed, both from the gross and microscopic aspects, calcific degenerative change or even advanced hyaline degeneration, was absent.

DISCUSSION

Studies based upon the use of venous autografts as replacements for diseased arteries are not new. Unsupported venous autografts of the thoracic aorta of both adult dogs and growing pigs are known to undergo dilatation in a significant number of instances. Sako (1951) believed that such dilatation may occur soon after the implantation of the graft. Our earlier findings would tend to substantiate this idea in some instances, Zech, Nyhus, Kanar, Schmitz, Sauvage, Moore, Merendino, and Harkins (1954).

Clinically, venous autografts of the extremities when supported by musculofascial structures are often successful and dilatation is not often encountered, Seeley, Hughes, Jahnke (1953). This knowledge, and the earlier work of Neuhof (1918), have undoubtedly served as a stimulus for finding a suitable buttress for venous autografts of the aorta where no musculofascial tissues are readily available.

Dilatation of venous autografts of the thoracic aorta has been restricted by the application of fascia lata sleeves about the implant, Sako (1951). Fibrous tissue irritants have long been employed in attempts at restricting the activities of arterial aneurysms. The results have been variable, but there appears to be

merit to such procedures, Zech, Merendino (1954). We have previously reported our results with the topical application of crystalline dicetyl phosphate about venous autografts of the thoracic aorta of growing pigs. These results suggested that if this substance was adequately applied, a fibrous tissue reaction of sufficient magnitude to restrict delayed dilatation of the graft would occur; however, it does not appear that this substance restricts the immediate dilatation of the grafts.

Woodhall and Golden (1953), in studies based upon the fibroplastic response of various tissue irritants placed about the cerebral arteries of rabbits, found that hevea latex produced an early, evenly distributed, adherent and moderately intense inflammatory reaction about the arteries in question in 7 days, and that at 21 days the tissue was dense and firm. Furthermore, these authors reported that this substance did not appear to affect the function of the vessel despite a definite thickening of the arterial walls.

In essence, our findings indicate that hevea latex as employed does incite a rather diffuse fibroblastic reaction of moderate degree. All graft walls so treated were thickened and one was even occluded by a combination of organized thrombus and mural thickening. In spite of these occurrences, dilatations did occur; these were generally diffuse with superimposed daughter aneurysms. This may be the result of inadequate topical application of latex, but this is not probable. In addition we noted a limitation of linear graft growth (table I). The significance of this failure of linear growth is not known.

Attempts at reducing venous caliber by plication have been previously reported, Schmitz, Sauvage, Kanar, and Harkins (1953). It has been noted subsequently in our laboratory that a few untreated fresh venous autografts had perfect dimensional changes at the time of harvest, and that in each instance the graft at implantation had been about 25 per cent smaller in external diameter than the recipient host aorta. These present studies have subjected grafts with an average external diameter of 71 per cent (produced by plication) of the adjacent aorta to the severe stress of rapid growth of the graft in the thoracic aorta. Plication did not appear an adequate means of preventing dilatation of the graft, although the dimensional changes incumbent upon growth are more nearly normal than when latex was used. Aneurysms present were smaller and less frequent than when plication was employed. This procedure undoubtedly is of value in reducing the size of venous autografts if such are to be used. We did not encounter any graft occlusions or mural thrombus formation, a fact which might indicate that the procedure need not carry an undue risk of thrombosis.

SUMMARY

Thirteen venous autografts of the thoracic aorta of growing pigs were either bathed in liquid latex after implantation or had their caliber reduced by the process of graft plication. There were 2 occluded grafts, both of which had been treated with latex; the remaining grafts were patent. All grafts underwent dilatation, and daughter aneurysms were frequently encountered. Latex treated grafts underwent more extensive dilatation and less linear growth than did the plicated grafts. Latex appears to promote a rather definite symmetric adventitial fibrosis without noticeably altering the graft. Neither procedure employed ap-

pears to be a satisfactory method for preventing the dilatation of venous autografts of the thoracic aorta of growing pigs.

CONCLUSIONS

Liquid hevea latex is a fibroblastic agent, but the fibrous tissue formed does not appear to prevent dilatation of venous autografts of the thoracic aorta of growing pigs.

Plication of venous thoracic autografts can be accomplished without undue risk of thrombosis, but such a graft will still undergo dilatation and aneurysm formation.

Latex may prevent linear graft growth, but it does not affect the animals as a whole.

Univ. of Wash.
School of Med.
Seattle 5, Wash.

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ABRONCHIECTATIC BRONCHIECTASIS: THE RECOGNITION OF SURGICAL PNEUMONITIS

RODGER E. MACQUIGG, M.D.

Albuquerque, New Mexico

Identification of bronchiectasis has been dated to Laennec's publication in 1819[3], but real appreciation of its incidence and precise correlation with symptoms and therapy awaited bronchography. Use of bismuth powder insufflation, as described by Jackson 100 years later[2], unfortunately usually gives poor filling of the terminal branches where the significant pathology occurs, although we still use this technic occasionally because of the excellent delineation of the entire surface of the trachea or major bronchi. Lipiodol was introduced in 1922 and with the development of safe resection technics giving stimulus to study of the disease, there now seems to be reasonable uniformity of opinion in progressive centers as to the indication for resection depending on the distribution, the symptoms, and the condition of the patient, all of which need not be further discussed here.

This paper is designed to call more general attention to the fact that the actual gross ectasia of the bronchi, which naturally first attracted notice of the pathologist and later of the roentgenologist, is by no means an essential feature of this disease from the standpoint of the patient whose health is to be restored and preserved. An inevitable sequel to modern compartmentation of clinical work is the dependence of the specialist upon men in widely diverse fields for his case finding. It is of little use for him to work out identification of a disease and precise technics for its cure if it is unrecognized by many practitioners to whom the patient turns.

We were all given the picture in our student days of the bronchiectatic patient with the productive cough. Yet bronchiectasis sicca, for example, has long been known, although this form of the disease is often dismissed as a *"cigarette cough"* or *"chronic bronchitis."* Since the basal bronchi are often examined at autopsy in such a fashion that involved segments may well be overlooked, the true incidence in most areas is quite unknown. We can certainly state that many an individual has proceeded ultimately to develop pulmonary fibrosis and emphysema with prolonged crippling and final death from right heart failure or pneumonia because such foci have been unrecognized until so much damage has been wrought that investigation such as bronchography was obviously academic. It is interesting that dry bronchiectasis seems to have a greater incidence of hemoptysis than does the wet type for reasons that are obscure.[1]

Granted that production of notable sputum is therefore by no means inherent in the disease, let us consider the ectasia itself. It is undeniable that a considerable number of patients can be shown to have surprisingly advanced ectasia with

Head of the Section on Thoracic Surgery, Lovelace Clinic, Albuquerque, New Mexico.
Presented during the annual assembly of The Southwestern Surgical Congress, Kansas City, Missouri, Sept. 5–7, 1955.

no apparent symptoms from the deformity. Many of these people do have, with intercurrent respiratory infections, episodes of sputum production, yet may escape frank pneumonitis or any evidence of chronic toxicity such as fever or debility. The bronchiectasis following tuberculosis furnishes an example, particularly if drained adequately because of its location.

Before considering the reverse proposition, that an undetermined number of individuals have significant symptomatic disease without any demonstrable ectasia, let us briefly assess localized bacterial infection as a factor in the chronicity and recurrence of pneumonitis. Conclusive evidence against necessity for the action of bacteria in perpetuation of chronic pneumonitis appears to be furnished by our present knowledge of the postpneumonic granuloma. For those unfamiliar with this condition, it may be explained that patients are appearing with symptoms resembling those under discussion, but whose roentgenograms reveal a *mass*, usually dating from a pneumonic episode and simulating in many cases the shadow of a malignancy. After resection, as several authors have noted, these patients are cured and yet the most careful culture studies may reveal no pyogens or other microbial agents to explain the existence of symptoms. Waddell and others have published brilliant papers[4, 5] explaining some of this mystery by finding cholesterol deposits in these granulomas, with associated chronic irritation as evinced by round cell infiltration. Some of our most typical material was carefully searched with the proper stains for similar deposits hoping to extend this finding to explain the recurrence or persistence of symptomatic areas where *no actual granuloma had formed nor demonstrable ectasia* such as reported in their cases. These deposits were not found, although suitable material should be further examined to settle this point.

All this leaves us then with the case where there has been clinical and roentgenologic evidence of pneumonitis recurring in one or more areas and in which medical therapy is clearly insufficient and yet no surgery has been offered in the past because there were none of the usually accepted entities identifiable as justification for resection. Some striking illustrations of this syndrome may be briefly presented.

CASE REPORTS

A 57 year old widow appeared in the spring of 1952 complaining of recurrent pneumonitis over the previous three years. Systems review revealed a draining ear dating from childhood and there was some degenerative arthritis. In February 1953, her physician, Doctor C. M. Kemper, recorded that since the previous May he had treated her for recurrent pneumonitis in the right lower lobe no less than seven times with pleuritic pain and temperatures to 103 or 104 F. (fig. 1, 2). A bronchogram on Jan. 2, 1953, showed no bronchiectasis but some obstructive emphysema (fig. 3). He also noted that with each succeeding attack it took longer to obtain relief with the various antibiotics used. Bronchoscopy on Feb. 9, 1953, revealed only some chronic edema in the right base. On February 17 the basal segments of the right lower lobe were resected. Our pathologist, Doctor T. L. Chiffelle, found gray-yellow mucus containing many polys in the bronchi, but no ectasia anywhere. There was, however, squamous metaplasia of the bronchial epithelium and mild chronic bronchitis.

Three months later some musical rales were still heard in all areas, but the patient was definitely improved. I advised her to inhale plain steam p.r.n. and to take vitamin supplements including A and D, with courses of tetracycline, for intercurrent respiratory infections, on the impression that she might have a general tendency to bronchiolar metaplasia

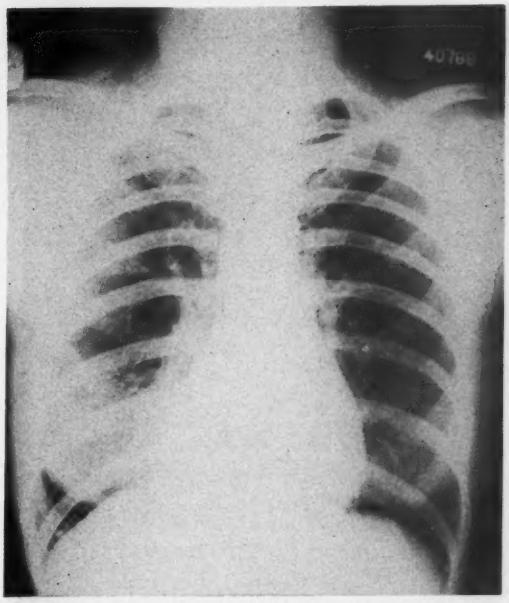

FIG. 1. Roentgenogram taken January 1950, showing right basal pneumonitis

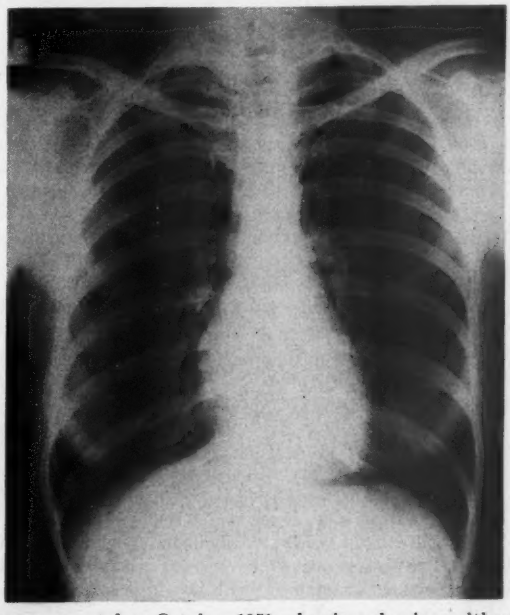

FIG. 2. Roentgenogram taken October 1951, showing clearing, although patient still symptomatic.

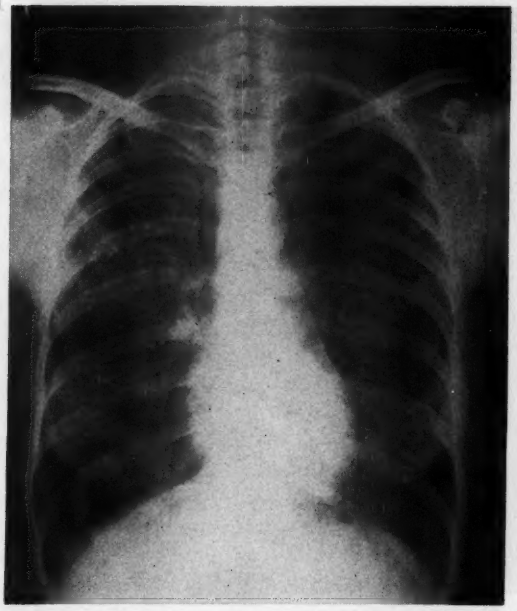

FIG. 3. Roentgenogram taken 24 hours after bronchography in January 1953 shows "arborization" still defective in right base, although no bronchiectasis was found.

and tenacious secretions. Her subsequent course has been very satisfactory although a year later she had a period of mucus production with coarse rhonchi, clearing again on tetracycline, 250 milligrams every 6 hours for 10 days.

In figure 4 we see a section from the periphery of the involved area showing normal ciliated mucosa and in figure 5 we see one of the innumerable metaplastic bronchioles with loss of the cilia and marked round cell infiltration. We might comment that it was a difficult decision to offer surgery to this patient with such vague localization and so little pertinent material available in the literature. But the proof of the pudding was in the eating as she feels definitely stronger as well as escaping from the increasingly frequent episodes of manifest pneumonitis.

Another case was that of a 55 year old lady whom I saw 2 years ago complaining of pleurisy attacks since age 45, beginning at intervals of 4 or 5 months, but increasingly frequent. At age 53 an intractable dry cough developed, more severe on reclining or even on bending. In January 1953, she was hospitalized for 6 days with pneumonitis and fever and since then had developed a fever to 101.4 or 101.6 F. and lasting for 3 or 4 days every 2 or 3 weeks. Penicillin appeared to accelerate recovery from most of these attacks. There was no history of asthma, hay fever, or dyspnea. A small area of tubular breath sounds was noted over the right middle lobe. As late as August 1953 roentgenograms were not diagnostic (fig. 6). Bronchograms done in August 1953 showed poor bronchiolar filling in the right base, but no bronchiectasis. On October 3, I excised the right middle lobe and an adjacent strip of the anterior segment of the right upper lobe, as the medial segment of the right middle lobe was found chronically atelectatic and fibrotic and the immediately adjacent right upper lobe was liver-like in consistency. The postoperative recovery was satisfactory and subsequently the patient was able to work without constant interruption by respiratory illness. She has had no fever for more than a year. Doctor Chiffelle reported the specimen as chronic and focal

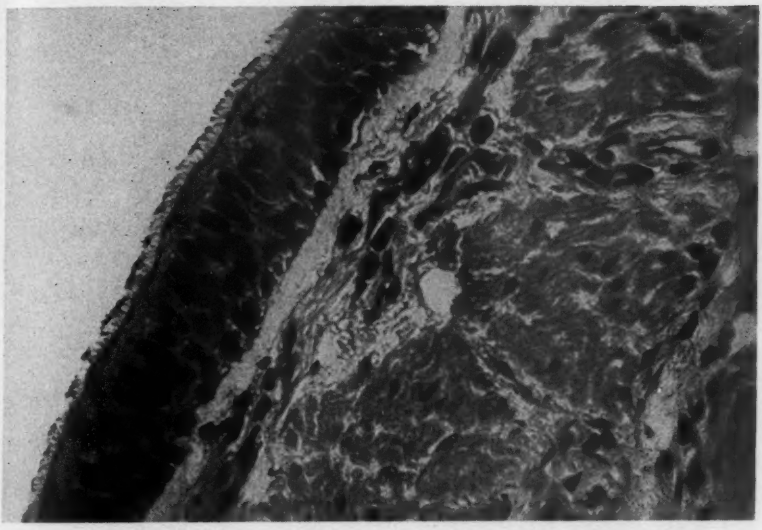

FIG. 4. Bronchiole with normal ciliated mucosa (in periphery of involved area)

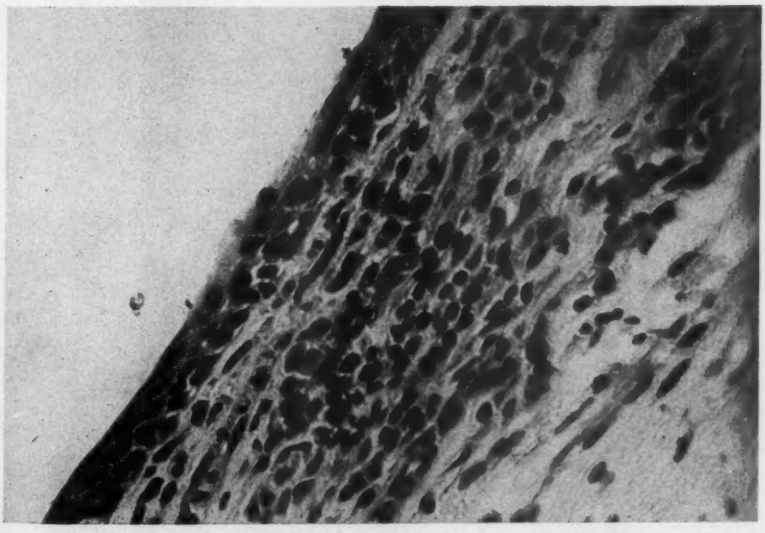

FIG. 5. Typically involved bronchiole with loss of cilia, round cell infiltration, and some fibroplasia.

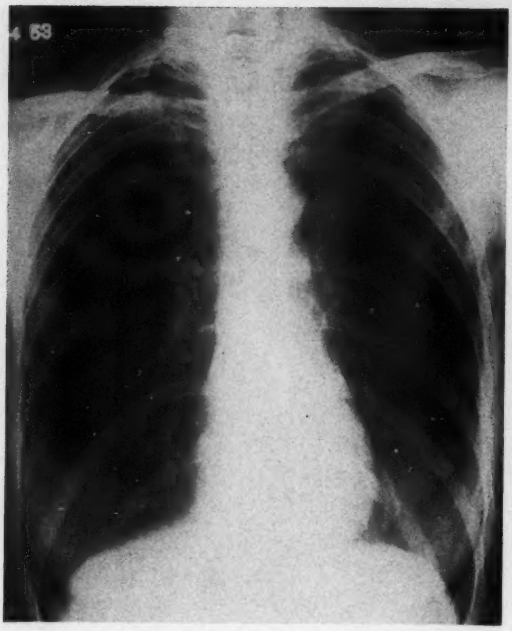

FIG. 6a

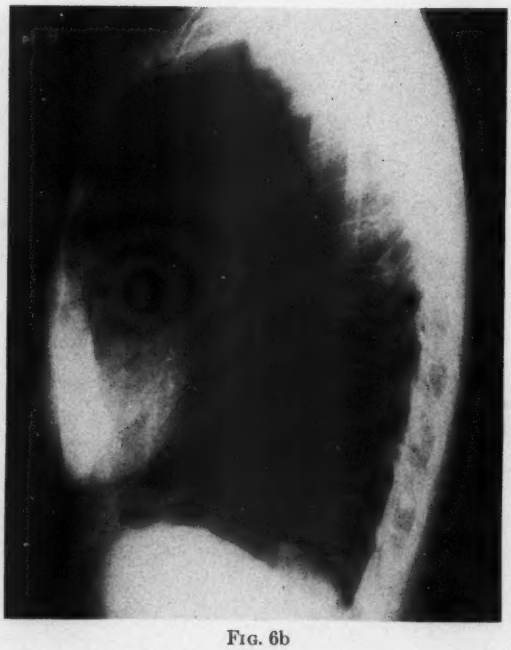

FIG. 6b

FIG. 6. Posteroanterior and lateral roentgenograms revealing no pneumonitis in August 1953, despite almost constant symptoms for 7 months.

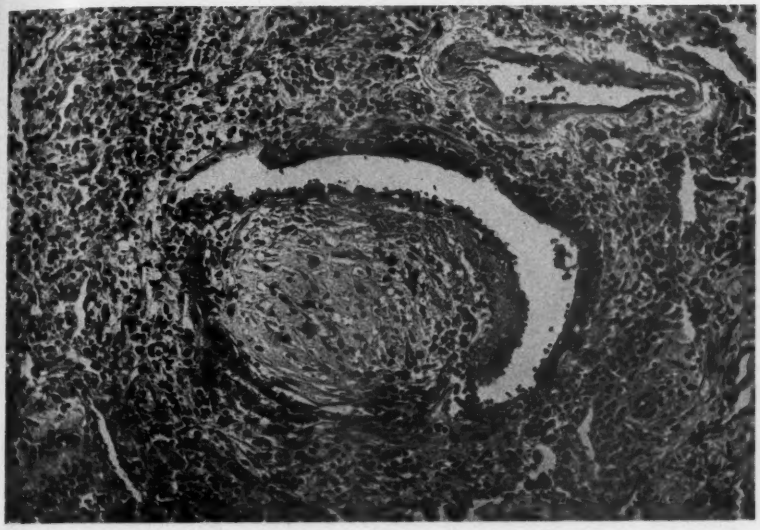

FIG. 7. Bronchiolar lumen almost occluded by edematous fibrous mass; note chronic inflammation.

acute pneumonitis with peribronchiolar fibrosis and round cell infiltration (figs. 7, 8). The inflammation was centered particularly about small bronchioles with formation of peculiar polypoid edematous fibrous masses which partially obstruct the lumen. Emphysema and focal atelectasis also were evident. Time does not permit showing other interesting cases of pneumonitis recurring in one or more closely adjacent areas and yet with good bronchograms showing no bronchiectasis, but this should suffice to outline our clinical syndrome.

We believe that the absence of ectasia may be partly explained by variations in individual fibrocytic response. Just as some tend to form keloids after incision, some may have a peribronchial fibrosis which keeps pace with the destruction of yellow elastic fibers so that actual ectasia never takes place, even in the smaller bronchi. However, with metaplasia and loss of cilia, stagnation of the ever present secretions, with symptomatic tiny areas of atelectasis and with all the other sequelae of the so-called obstructive syndrome, may result.

It is now our responsibility to recognize these cases and to select those patients in whom there is sufficient localization of the pneumonitis, to permit, if symptoms warrant, a definitive surgical therapy. It seems likely that we are seeing increasing numbers of these cases because before the antibiotic era frank bronchiectasis or abscess supervened. But the recurrence after supposedly adequate medical therapy and the toxicity between the acute episodes would indicate that antibiotics are inadequate. It seems possible that gradations will be found between this condition and the postpneumonic granulomas previously reported, but the lack of demonstrable ectasia or of lipid deposits, as well as minimal mass formation, implies a distinct disease process.

We might speak of the disease under discussion as an abronchiectatic bron-

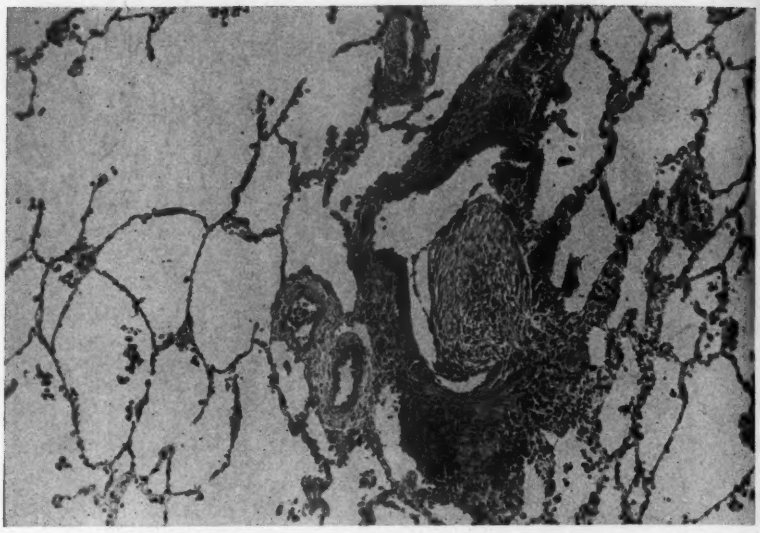

FIG. 8. Another of countless obstructing fibroplastic lesions; focal emphysema present

chiectasis. When the internists speak of an aleukemic leukemia, they imply that it is not the peripheral leukocytosis which cripples the patient, but rather the stifling overgrowth of the white cells in the marrow or other tissues which is the essential pathology. By the same token, we are seeking to stress the fundamental *bronchiolar metaplasia* so that adequate therapy will no longer be withheld on the basis that no *bronchial ectasia* is demonstrable or other change such as frank granuloma formation persisting on roentgenologic examination.

SUMMARY

In conclusion, we would define this condition as a chronic and often localized bronchitis or bronchiolitis with metaplasia and bronchiolar fibrosis characterized by recurrent pneumonitis and perhaps occasionally by granuloma formation, but characteristically failing to yield known pathogens on culture of the resected specimens. It is manifest clinically by chronic or recurrent fever, pleuritis and debility, and by cough, usually nonproductive. It usually is associated with pulmonary emphysema. Bronchial ectasia is not typically found on bronchography, although areas may be found with deficient filling even on roentgenologic films taken hours after instillation of the oil. Resection where feasible is strongly recommended as the therapy of choice.

Lovelace Clinic
4800 Gibson Blvd., SE
Albuquerque, N. Mex.

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TREATMENT OF GASTRIC POLYPOSIS

JAMES W. HENDRICK, M.D., F.R.C.S.

Tuscaloosa, Alabama

Gastric polyposis is a relatively rare disease. The diagnosis of gastric polyps was infrequently made until Heinz[8] (1911) demonstrated their presence by roentgenoscopic examination. Schindler[16] (1922) successfully observed gastric polyposis with the gastroscope. Since that time the disease has been more frequently diagnosed due to improvements in our diagnostic armamentarium. Brunn and Pearl[2] (1926) published comprehensive reports on the clinical and pathologic manifestations of the disease; brought the literature to date, and reported several cases of their own. Edwards and Brown[3] (1950) surveyed the literature relative to the frequency of malignancy developing in gastric polyps.

The disease is of clinical importance to the physicians, radiologist and surgeon since the diagnosis may be confused with chronic atrophic gastritis or gastric cancer on roentgenologic or gastroscopic examination. Polypoid adenomas of the stomach produce bleeding, ulceration and changes in the motor function of the stomach, and are precursors of gastric cancer.

Incidence and Etiology. The incidence of gastric polyps varies with the source of statistical material. Eusterman[4] found 1.3 per cent of all gastric tumors encountered surgically to be benign. Rigler and Ericksen[15], in a survey of 6,742 consecutive autopsies, encountered 194 gastric tumors of which 50 (27 per cent) were benign. These authors reported that of 239 gastric neoplasms diagnosed roentgenologically, 26 (11 per cent) were benign. Spriggs and Marxer[17] found 30 cases of gastric polyposis during 2000 roentgenographic examinations of the stomach.

Investigators[2, 13] have observed the frequent association of gastric polyposis occurring in patients with achlorhydria and chronic hypertrophic gastritis. Hay,[7] reporting from the Cancer Detection Center at the University of Minnesota found 35 patients with gastric polyps in 1330 achlorhydric patients; of 50 patients with polyposis, 48 had severe atrophic gastritis; 5 of 115 pernicious anemia patients had gastric polyps. Other causative factors reported include syphilis, vitamin deficiency, intestinal parasites, and the unrestricted use of alcohol. The author, during the period from 1933 to 1955, observed 5 patients with gastric polyposis in 562 consecutive operations on the stomach and duodenum.

Gastric polyposis occurs more frequently in an older age group; 60 per cent are detected in the sixth and seventh decades. The ages of the 5 patients treated by the author varied from 50 to 74 years; 3 were men and 2 were women.

Pathology. The term gastric polyposis is used to designate any pedunculated, benign tumor arising from the mucous membrane of the stomach regardless of histologic structure. Adenomatous polyps may be single or multiple, pedunculated or sessile; they are sharply demarcated from the surrounding mucosa. Ulceration of a polyp occurs frequently but does not necessarily indicate malignancy. The stalk is composed of proliferated gastric epithelium with a connective tissue core[14].

Menetrier[12] (1888) described gastric polyposis and divided the disease into two groups: polyadenoma polypeaux, and the rarer type, polyadenoma en nappe. The former designated discrete polyps scattered over the mucosa of the stomach; the latter were well demarcated plaques of closely placed folds of hypertrophic mucous membrane. Brunn and Pearl[2] defined gastric polyposis by the presence of three or more polyps in the same stomach.

Polyps may occur in any area of the stomach; the majority are found in the antral region. It is difficult to determine the incidence of malignant degeneration of benign gastric polyps[3, 10, 11]. Polypoid cancers of the gastrointestinal tract are not uncommon but their origin is not necessarily in a benign epithelial polyp.

Symptoms and Signs. The symptoms of gastric polyps depends upon their size and location. Multiple, small gastric polyps may produce the same symptoms as chronic hypertrophic gastritis and/or achlorhydria or they may be asymptomatic. Many small benign gastric tumors are overlooked because of lack of definite symptoms. Three patients with multiple small polyps located in the distal half of the stomach complained of epigastric pain, gaseous indigestion, postprandial fullness and diarrhea with alternating constipation. Two patients had hematemesis. All 5 patients had a history of epigastric tenderness, weakness, weight loss, anorexia, dyspepsia, gastrointestinal bleeding, occult blood in the stools and anemia. A large polyp with a long pedicle located in the region of the pylorus can interfere with peristalsis of the stomach and produce pyloric obstruction or intussusception[6]. The obstruction may be of the ball-valve type and intermittent. Multiple polyps that are spread over a large area of the stomach frequently simulate carcinoma.

Diagnosis. Patients over 40 years of age with gastrointestinal symptoms that persist over a period of several weeks deserve a careful gastrointestinal study, i.e., gastric analysis, roentgenographic studies of the gallbladder, stomach, small bowel, colon and proctoscopic examination. If hypochromic anemia, achlorhydria and hyperplastic gastritis are present, polyposis should be considered[13]. Roentgenologic and gastroscopic examinations are the most reliable methods for detection of gastric polyps. Gastroscopic examination is valuable in the detection of polyps and differention between benign and malignant lesions[6].

TABLE I

Clinical manifestations of gastric polyposis

(Five cases)

	Total
Pain	5
Indigestion	5
Weight loss	5
Weakness	3
Anorexia	4
Occult blood in stools	5
Hematemesis	2
Anemia	5
Epigastric tenderness	5

TABLE II

Radiologic signs of gastric polyposis

(Five cases)

	Total
Filling defects	5
Irregular deformity	4
Mucosal changes	4
Loss of flexibility	4
Outlet obstruction	1
Negative shadows	5

The wide use of roentgenographic examination of patients with minimal gastric symptoms accounts for the increasing diagnosis of gastric polyps. Small polyps are detected most readily by the radiologist when only a small amount of barium is administered in the preliminary examination and pressure is exerted to produce a detailed mucosal pattern. When small gastric lesions are detected, the air contrast method is useful comparable to the use of this method for detection of polyps of the colon. Gastric polyps are demonstrated as negative shadows in the roentgenogram. The roentgenograms of 3 patients showed multiple negative shadows; the fourth showed a large negative shadow that was movable within the stomach which was diagnosed as either a bezoar or large polyp with a long stalk. The fifth patient had an irregular filling defect of the greater curvature, loss of flexibility of the gastric wall, and several negative shadows in other areas of the stomach which was polypoid carcinoma with many gastric polyps.

CASE REPORTS

Case 1: H. M., a white man, 61 years of age, complained of epigastric distress, belching, flatulence, and weakness over a period of 10 years, and a loss of 15 pounds in weight during the previous year. He had been placed on an ulcer management program for a period of 3 years without improvement. Examination revealed a well developed, poorly nourished white man. Erythrocyte count was 3,500,000 per cu. mm. with 10 grams of hemoglobin per 100 cc. Occult blood was present in the stools. Roentgenographic examination demonstrated the mucosal pattern of the stomach to be distorted with multiple negative shadows over the lower ⅔ (fig. 1). No free hydrochloric acid was present in the gastric analysis; total acidity was 8 units and 3 plus blood.

At operation diffuse gastric polyposis was found involving the lower ⅔ of the stomach. The involved area was soft and felt as though it contained a bag of worms. An anterior Hofmeister gastric resection was performed. The patient made an uneventful recovery; has been followed for a period of 13 years with no further gastric symptoms. The pathologic report was multiple benign adenomatous polyposis with atrophic gastritis (fig. 2).

Case 2: H. L. J., a white man 56 years of age, had a history of epigastric pain, recurrent episodes of nausea and vomiting of bright red blood at intervals for a period of 4 years. The patient had been hospitalized elsewhere on four occasions without relief of symptoms for partial or complete gastric obstruction supposedly due to a stenosing duodenal ulcer. Examination revealed a well developed, poorly nourished man. The erythrocyte count was 4,200,000 per cu. mm., hemoglobin 9 grams per 100 cc. Gastric analysis demonstrated a total acidity of 10 units with no free hydrochloric acid. Occult blood grade III was present in the stools. Roentgenographic examination of the stomach revealed a large atonic stomach with

FIG. 1. Case 1. Roentgenogram of the stomach demonstrating multiple gastric polyps shown as negative shadows. The lower half of the stomach contains numerous benign, adenomatous polyps, 2 to 3 cm. in height and 3 to 6 mm. in diameter.

a filling defect in the prepyloric region which was found by the radiologist during fluoroscopic examination to be moveable. It was thought the mass represented either bezoar or a gastric polyp with a long stalk. At operation a soft mobile tumor mass could be palpated in the stomach. Through a gastrotomy opening a soft, spongy polyp 8 cm. in diameter with a pedicle about 6 cm. in length was attached to the posterior wall of the stomach (fig. 3). The stalk of the polyp was 1 cm. in diameter and the base was 4 cm. in diameter. The base of the polyp was excised with the area of stomach wall. Pathologic examination revealed the adenomatous polyp, base and stalk to be benign. The patient made an uneventful recovery and has had no further gastric symptoms after a period of 8 years.

Case 3: N. R., a 57 year old white woman, complained of frequent episodes of epigastric distress, i.e., pain when the stomach was empty which was relieved by small amounts of food, anorexia, vomiting and weakness. She stated that her stools had been black on numerous occasions; she had been treated for hypochromic anemia over a period of 2 years. Examination revealed a poorly nourished white woman. The erythrocyte count was 3,100,000 per cu. mm. with 8 grams of hemoglobin per 100 cc. Gastric analysis did not reveal free hydrochloric acid; total acidity was 10 units and occult blood, grade III was present in the stools. Roentgenographic examination of the stomach revealed multiple small negative

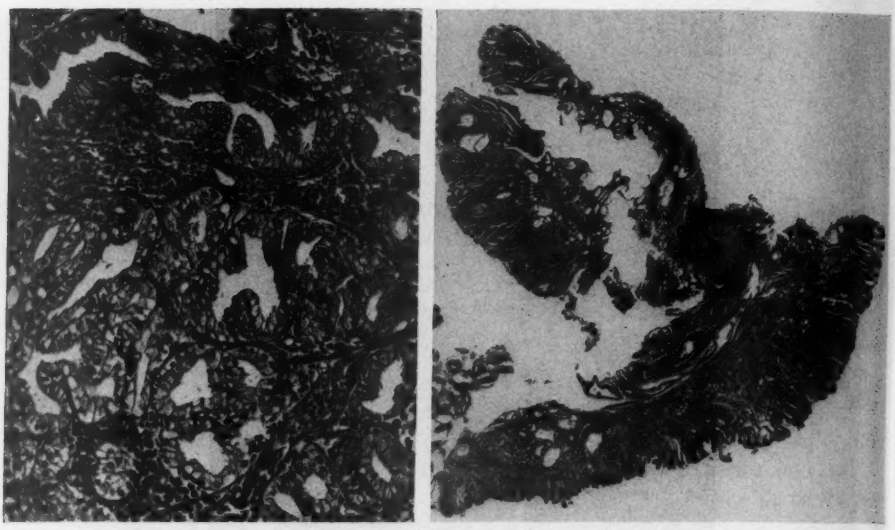

FIG. 2. Case 1. Photomicrograph demonstrating gastric mucous membrane with benign adenomatous polyps showing stalk of polyp and gastric glands forming the polyp.

FIG. 3. Case 2. Large adenomatous polyp 8 cm. in diameter with a stalk 6 cm. long attached to the posterior wall of the stomach. At intervals the polyp would protrude into the pylorus producing intermittent pyloric obstruction. The polyp, stalk and base were benign.

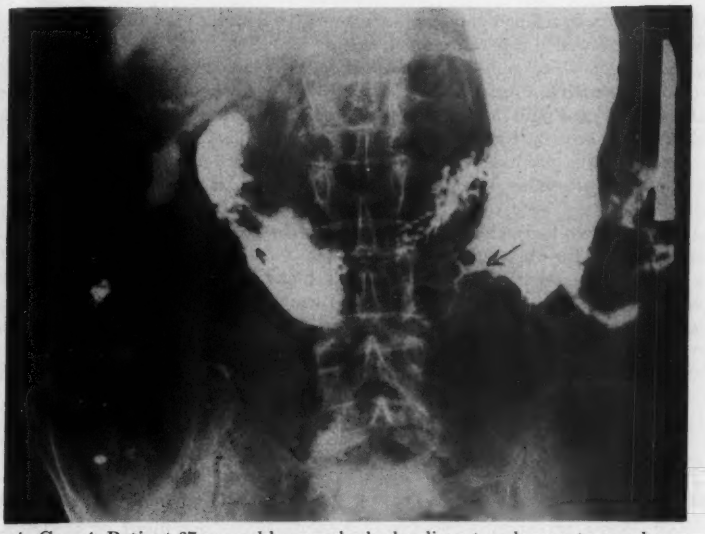

FIG. 4. Case 4. Patient 67 year old man who had a discrete adenomatous polyp removed from his stomach 8 years previously; the base of polyp had not been removed. Roentgenogram demonstrating polypoid carcinoma of the greater curvature of the stomach with numerous small polyps in surrounding area.

shadows beginning in the region of the pylorus and extending to involve the lower half of the stomach. A diagnosis of gastric polyposis was established. At operation the stomach felt normal but when it was opened the mucosa was involved by numerous polyps over its lower half. A Hofmeister type anterior resection was performed. Pathologic examination demonstrated numerous benign adenomatous polyps that varied in height from 6 mm. to 3 cm. and 3 to 6 mm. in diameter. The patient made an uneventful recovery with no further symptoms referable to the stomach. Four years later she developed symptoms of left colon cancer and was found to have an annular carcinoma of the sigmoid. An anterior resection was performed. The lymph nodes were negative for metastasis. The patient has been symptom free from both diseases for 5 years.

Case 4: C. W. W., a white man, 67 years of age, had a history of benign polypoid growth which had been removed from the stomach 8 years previously. He was free from gastric distress for 5 years after which he began to have pain in the epigastric area, weakness, nausea and loss of weight. He had been placed on ulcer management by his physician without relief of symptoms and was referred to the author for treatment. The tumor removed previously was described as a large gastric polyp but had not been submitted for pathological examination. Examination revealed erythrocyte count of 3,000,000 per cu. mm. with a hemoglobin of 8 grams per 100 cc. No free hydrochloric acid was present in gastric analysis; 4 plus blood was found. Occult blood was present in the stools. Roentgenographic examination demonstrated an irregular filling defect along the greater curvature of the stomach; a diagnosis of carcinoma was made (fig. 4). At operation a moveable tumor was found involving the lower half of the greater curvature of the stomach; there were no enlarged lymph nodes in the drainage area. An anterior Hofmeister gastrectomy was performed with removal of $^{9}/_{10}$ of the stomach, omentum and spleen. The patient made an uneventful recovery from the operative procedure. The pathologic specimen contained polypoid type carcinoma with numerous small polyps $^{1}/_{2}$ to 1 cm. in diameter surrounding the carcinoma. The serosa was not invaded and the lymph nodes were negative. The patient was symptom free for 3 years

after which he then developed painless jaundice with a nodular mass in the liver. Exploratory laparotomy revealed generalized metastases.

Case 5: H. G., a white woman, 74 years of age, complained of epigastric distress over a period of 12 years with anorexia, nausea, and on occasions, vomiting with hematemesis. She complained of a vague pain in the epigastric region, postprandial fullness and diarrhea at intervals. She had been on various types of diets, and medication for stomach trouble without improvement; she also had been treated for anemia for 4 years. Examination revealed a well developed, poorly nourished white woman with a weight loss of 20 pounds during the previous year. The erythrocyte count was 3,000,000 per cu. mm. hemoglobin 9 grams per 100 cc. There was no free hydrochloric acid present in the gastric analysis. Occult blood was found in the stools. Roentgenographic examination of the stomach revealed multiple filling defects along the greater curvature of the stomach and in the pyloric area. A diagnosis of multiple polyposis was made. At operation the stomach had a normal appearance; no polyp could be palpated through the intact gastric walls. The stomach was opened and found to contain numerous small polyps involving the lower ⅔. An anterior Hofmeister partial gastrectomy was performed. The patient made an uneventful recovery and has been symptom free for a period of 1 year. The hypochromic anemia has corrected itself. Pathologic diagnosis was multiple benign adenomatous polyps of the stomach.

TREATMENT

There are differences of opinion regarding the treatment of gastric polyposis[6, 7, 17]. Some physicians recommend medical management of patients who are asymptomatic or mildly symptomatic, with polyps less than 1 or 2 cm. in diameter which appear benign on roentgenographic and gastroscopic examination[7]. The advocates of this therapy prefer to observe such patients every 3 or 4 months during the first year and biannually thereafter; if severe symptoms develop, i.e., bleeding, loss of weight or evidence of malignancy, operative treatment is instituted.

Subtotal gastric resection is the method utilized by the author in treatment of patients with gastric polyposis, except in those who have a large discrete polyp with a long pedicle, in which case the base and the surrounding area of the stomach wall is excised. In such cases the polyp and its base is submitted for immediate frozen section to determine if malignancy is present. Patients with multiple polyposis accompanied by achlorhydria and chronic gastritis, with or without mucosal atrophy, are considered potential candidates for malignant transformation. Subtotal gastric resection[1, 3, 10, 11] is the most feasible manner to remove such adenomatous polyps and adjacent atrophic mucosa to prevent the further development of adenomas and carcinoma. The author has not observed gastric polyps of the cardia but in each case the entire interior of the stomach is inspected at the time of operation.

SUMMARY

Gastric polyposis is an infrequent surgical disease. The symptoms, which depend on the size and location of the polyp, are indigestion, postprandial fullness, anemia, and loss of weight and strength. Roentgenographic and gastroscopic examinations are the most reliable methods for detection of gastric polyps. Chronic atrophic gastritis and achlorhydria frequently are associated with gastric polyposis. Subtotal gastric resection is the treatment of choice, except in

patients with a large discrete polyp, in which case, excision of the polyp, its base and an area of the stomach wall around the base is recommended.

602 First National Bank Bldg.
Tuscaloosa, Alabama

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MALIGNANT DEGENERATION IN SEBACEOUS CYSTS

HERMAN CHARACHE, M.D.

Brooklyn, New York

No tumor has received so little attention and been treated with so much contempt as the sebaceous cyst. The majority of them are removed in the doctor's office and relegated to the waste can. Some are removed in clinics and seldom reach the pathology laboratory. A few are removed in the operating room, but not many of these are examined histologically.

Ninety-six cases of sebaceous cysts with malignant degeneration were found recorded in the literature up to the present time. The majority were single case reports. The largest series of cases were reported by Caylor[2], Bishop[1], and Stone and Abbey[6]. They reported a total of 718 cases of sebaceous cysts of which 31 were malignant, an incidence of 4.3 per cent. How many sebaceous cysts with malignant degeneration have been discarded undiagnosed will never be known. When they are diagnosed after a recurrence, the diagnosis usually is "*squamous cell carcinoma of the skin.*" Nor can we estimate the number of cases of histologically proved malignant degeneration of sebaceous cysts that are never reported.

A clinical study of the 96 cases of sebaceous cysts with malignant degeneration reveals that male and female were equally affected. The majority of the patients were in the fifth decade or older. The tendency for the lesion to become malignant increases as the patient grows older[6]. The youngest patient was 16 years old[5], the oldest 83[2]. One patient was a Negro[1]. The sites of predilection are the head and neck. Histologically, squamous cell carcinoma was the most common diagnosis, occurring in over 90 per cent of the patients. The remainder were of the basal cell type. The former type was composed mainly of fairly well differentiated cells with considerable hornification, pearls and spines. A history of long duration and recurrences was the rule. In many patients ulceration preceded the discovery of the tumor. Metastasis was infrequent. Three cases with general metastasis were reported by Caylor[2], Seff and Berkowitz[4], and Gregersenn[3]. Gregersenn's case metastasized to the patient's brain. The tumor grows slowly within the cyst wall. Invasion of the surrounding structures is a late occurrence.

The treatment of choice is complete excision of all sebaceous cysts before they become malignant. A histologic study of all removed cysts should be made. In recurrent cases wide excision is particularly recommended instead of enucleation which is so commonly practised. If they are reported as malignant postoperatively, radiation therapy should be instituted.

The prognosis is good if the lesion is removed early before it involves the surrounding tissues.

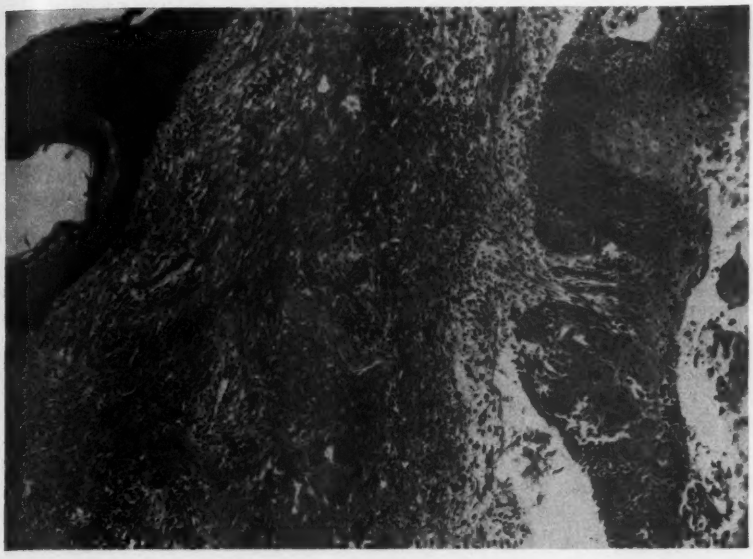

FIG. 1. Photomicrograph (100 ×) showing section of sebaceous cyst lined by stratified squamous epithelium with incipient malignant changes, characterized by the formation of epithelial pearls and variations in size, shape and staining reaction.

CASE REPORT

A 31 year old single white woman was seen in the office on April 16, 1952 complaining of a *"boil"* on the right arm. About two weeks before she had noticed a *"pimple"* on the right arm which changed to a *"boil"* after she squeezed it.

She had always been in good health. On Feb. 20, 1946 she had a sebaceous cyst removed from her forehead which was diagnosed histologically as benign.

Examination showed the patient to be in good physical condition. A small cystic mass about 2 cm. in diameter was found on the right arm. The overlying skin was partially ulcerated. A diagnosis of sebaceous cyst was made and she was admitted to Prospect Heights Hospital, where it was excised in the operating room. She was discharged the following day. The histologic diagnosis was *"sebaceous cyst with incipient intraepidermic epithelioma."* The wound healed by primary intention, and she received postoperative radiotherapy. There was no recurrence on April 30, 1955, and the patient is in good physical condition.

SUMMARY

Very little attention is paid to sebaceous cysts. The majority of them are removed without histologic examination of the tissue.

Ninety-six cases of sebaceous cysts with malignant degeneration were found recorded in the literature up to the present writing. Of 718 cases of sebaceous cyst examined microscopically[1, 2, 6] 4.3 per cent were malignant.

The clinical and pathologic findings recorded in the literature are discussed.

An example of malignant degeneration of a sebaceous cyst, with photomicrograph, is reported.

75 Prospect Park Southwest
Brooklyn 15, N. Y.

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TWENTY-FIVE YEARS OF PROGRESS IN THE TREATMENT OF FRACTURES

MARCUS J. STEWART, M.D.

Memphis, Tenn.

It is gratifying to note that in the past quarter of a century all branches of the medical profession have surged forward with tireless effort in their own respective fields of endeavor. The anatomist, chemist, physiologist and physician are becoming more amalgamated into a team dedicated to the advancement of medical science.

Twenty-five years ago Dr. Willis Campbell[5] addressed this Society on "Ununited Fractures". He pointed out that five types of bone transplants had been employed in the treatment of ununited fractures; namely, (1) medullary, (2) osteoperiosteal, (3) chip, (4) inlay, and (5) onlay. He championed the onlay graft, and some of the best results in all the annals of orthopaedic surgery in the treatment of ununited fractures are those patients who came under his hand. His review before this group of the physiology of bone healing and repair was outstanding and most of the principles expounded at that time remain unchanged. Although these principles of physiology have stood the test of time, the technics in orthopaedic surgery have made great strides.

Antibiotics

One of the greatest advances in the treatment of ununited fractures, which are so often complicated by infection, has been the introduction of modern antibiotic therapy. One of Campbell's[6] last contributions was the analysis and review of the effect of sulfanilamide in the treatment of compound fractures. The discovery and development of penicillin by Drs. Fleming and Flories truly opened the doors to the field of modern antibiotic therapy and now the great scientists and laboratories of the world give us many useful drugs with astounding bacteriostatic and bacteriocidal properties.

Anesthesiology

Anesthesiology has made such strides in the past 25 years that it is now possible for the patient to rest in safety while the surgeon does prolonged and extensive operations for the restoration of bone continuity and strength.

Metallurgy

The developments in metallurgy in the past two decades have been remarkable. We have progressed from the old rusting, corroding vanadium steel to the electrolitically silent, vitallium and S.M.O. stainless steel. These metals can be placed in the body tissues with impunity, even if there is impending infection.

Presented during the Atlanta assembly of The Southeastern Surgical Congress, Feb. 22, 1955.

Intramedullary fixation of long bones, although used and publicized as early as the war between the states, was not put to practical use until World War II, when Kuntscher[15, 16] popularized his intramedullary fixation for fracture of the femur and other long bones. This is not only of great benefit in the treatment of large and difficult ununited fractures but is of great assistance in the prevention of nonunion. The use of intramedullary fixation and cancellous bone with the so-called Phemister[21] grafts for nonunion has become a popular and dependable procedure. This inert intramedullary splinting gives immobilization while cancellous bone placed at the fracture site furnishes a calcific surcharge to the parent bone, assuring, as a rule, amalgamation into a solid union.

Bone Bank

The advent of the bone bank has made possible the fixation of ununited fractures without the added burden of a double operation or the expense of other parts of the body for donation of bone. Also, by this technic, the operative time has been greatly diminished. Bone-bank bone and freeze dried bone has proved a great asset in the treatment of fractures, both fresh and old[8].

Transfusions

In the past 25 years the utilization of whole blood and plasma has reached an efficient and practical use never before visualized. It would be hard for our forefathers to have conceived that fresh human blood could be transported thousands of miles in a few days and become life saving on the front line of battle. Transfusions have become as commonplace in the operating room during extensive surgery as the hypodermic for the relief of pain was 25 years ago.

Plastic Surgery

Our colleagues in plastic surgery have made great strides in skin grafting of varied types, particularly in covering large soft tissue and bony defects. This protection of the bone from contamination, plus the restoration of a subcutaneous vascular bed, is responsible for many good results where previously nonunion was inevitable.

Mobilization

World War II furnished the great checkrein for the pendulum of immobilization and for popularizing the practice of "*mobilization not immobilization*". Rigid immobilization, "*until all fractures are radiographically solid*", has been found unnecessary in many instances; especially is this true when the bone has an abundant blood supply. Fractures of the ribs, clavicle, facial and pelvic bones heal rapidly and without nonunion. Fractures of the femur treated by skeletal traction heal much faster than those immobilized in plaster or operated upon and fixed with plates and screws. Most fractures of the humerus do not require rigid immobilization, but this is a difficult fact to teach certain schools of surgery.[24] Campbell[5, 7] commented on several occasions, to wit, "*the hanging cast is effective in treatment of fracture of the humerus but the method is physiologically inconsistent.*"

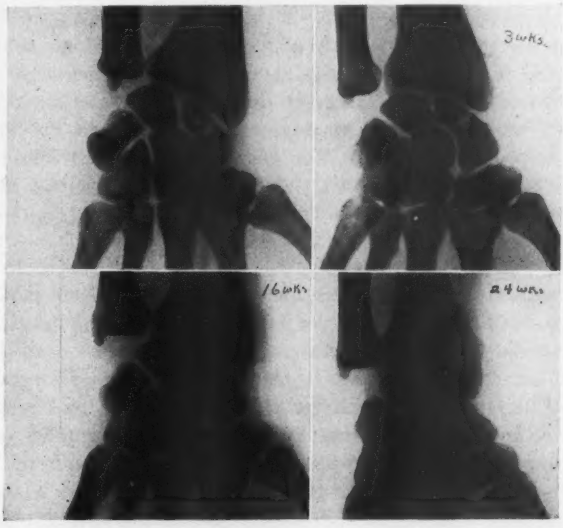

FIG. 1. Note fracture in the waist of the carpo-navicular which was undiagnosed for 15 weeks, then immobilized in plaster for 24 weeks; 2 weeks after removal of plaster the range of motion was normal in the hand and wrist.

The hanging cast when properly employed has removed fractures of the humerus from one of the leading contenders for the production of nonunion to one of the rarest sites for its development.

During World War II it was re-emphasized that prolonged immobilization of the carpal navicular is the treatment of choice for this fracture, provided mobilization of the fingers and their metacarpophalangeal joints is maintained[25] (fig. 1).

During the last decade early active exercise with early ambulation has become common practice. The great strides in internal fixation, including intramedullary immobilization, have made this phase of progress possible. During the Second World War rehabilitation gave birth to a new enthusiasm for early muscular exercise and for mobilization of the bedridden patient. Out of this, on June 12, 1944, was born a new division of the United States Army Medical Corps, "The Three Thousand Bed Convalescent Center," which has become the stepping stone from bedridden hospitalization back to active duty. Literally thousands of patients were returned to the fighting fronts by means of this concept of conservation and restoration. These centers were first organized and developed under the guidance of the orthopaedic surgeons of the Army in the European Theater. Then it spread rapidly and effectively to the Zone of Interior and to each branch of the Military Medical Service. There also was born the profession of Corrective Therapy, not as a counterpart but as a team-partner to Physical Therapy[23].

Replacement Therapy

The introduction of inert metals and alloys proved a great boon to the development of internal prosthetic, or replacement therapy, particularly in ununited fractures of the femoral neck. The pendulum swung far to the left in this form of treatment, but research, review, study and logical thinking are bringing this medium into its proper perspective. You may now have an artificial replacement for your carpal navicular, radial head, tibial plateau, patella, elbow joint, femoral head and many other bones of the living body.

Vascular Surgery

It is a well established fact that no fracture will heal without adequate blood supply. Many methods have been devised to maintain and re-establish blood supply to a specific part of the body in an attempt to insure healing of wounds and fractures. During and since the Korean conflict, vascular surgery has made important and heroic strides. In the past few years many lives and extremities have been saved by modern *"blood vessel surgery."* The horizon has been opened, and in the years to come nothing will do so much to prevent nonunion of certain fractures as re-establishment of the vascular tree.

As in all branches of medicine and surgery, the most important single feature in treatment is prophylaxis, and in this field the management of fractures has made its greatest strides, that is the prevention of nonunion.

MODERN CONCEPT OF FRACTURE HEALING

It is imperative that an adequate hematoma be formed between the fractured bones to insure the development of a calcifiable matrix. During the first week after fracture there is a concentration of calcium and phosphorus in the hematoma. This comes primarily from the bone ends where absorption may be detected by roentgenogram; it occurs as the result of hyperemia or vasodilation due to the histamine and acetylcholine effect upon the bone ends at the fracture site. There is a rapid increase in phosphatase, 6 to 7 times normal, which comes from the action of the osteoblast and cartilage cells. Phosphatase is produced by hydrolysis of the organically bound phosphoric acid of the plasma which results in a saturation with calcium phosphatase. This continues on an average for 2 to 2½ months, depending upon the age of the patients and site of fracture. The so-called acid tide which occurs at the fracture site exists for possibly 2 weeks when it gives away to a swing of the pH back to the alkaline side.

Sir Reginald Watson-Jones[28] stated, *"there is only one cause of nonunion of fractures with a continuous hematoma between the fragments—the cause of nonunion is inadequate immobilization."* This does not necessarily mean rigid immobilization for we cannot agree with the old theories of the nineteenth century when Hugh Owen Thomas stated, *"we do not expect union of bone if motion of the fragments is permitted;"* or Richard von Voltman who stated at about the same time, *"the speed of reunion of bone is in direct ratio to the rigidity with which the two pieces are placed together."* If there is an adequate blood supply, so that nature can properly mobilize her healing properties a little motion often accelerates the rate of healing.

This was pointed out by Leriche and Policard[17] in their treatise on bone physiology.

Urist, Mazet, and McLean[27] have recently championed a theory that bone formation always begins in the fracture site by the proliferation of the periosteum and endosteum at some distance from the line of fracture and that it progresses by extension of these properties. They state that periosteal reaction is intramembranous ossification, while the endosteal reaction is appositional bone production. However, in both instances the new bone originates from cells which are direct descendents of the osteoblast. If there is a gap to be bridged between bone ends, then there is a formation of fibrocartilaginous tissue which apparently originates from the same source plus an assist from the reticulum cells of the bone marrow. Some minimal amount of fibrocartilaginous tissue is developed at the fracture site even in the presence of compression. Thus, the formation in this primitive tissue is similar to that of fetal osteogenesis. New bone grows into the fracture callus and replaces this fibrocartilaginous tissue. The production of nonunion, according to Urist and his colleagues results from fibrinoid degeneration in the interior of this fibrocartilaginous callus and failure to establish an effective induction system for osteogenesis to proceed across the fracture line. These authors believe that nonunion per se is exceedingly rare unless mechanically produced. They state that the minimal healing time which should elapse before nonunion is declared, should be 12 months for noncomminuted fractures, 18 months for comminuted fractures with single wedge fragments, and 24 months for severely comminuted or displaced segmental fractures. Even though this may theoretically be true, it does not represent a practical basis for the treatment of fractures.

Nonunion in long bones is often associated with this fibrinoid degeneration at the fracture site. Degeneration of the fibrous tissue or fibrocartilage at the fracture is, as a rule, due to inadequate blood supply, and inadequate immobilization with poor healing and may lead to a true pseudarthrosis. The myxomatous degeneration and mucin formation at this false joint is similar to that which occurs in an adventitious bursa. Fibrinoid degeneration has been known since 1880 when Neuman[19] pointed out the traumatic inflammatory conditions which may lead to its formation. Urist stated that, morphologically fibrinoid tissue appears as a mass consisting of collagen and ground substance in all stages of degeneration. Fibrinoid has been produced experimentally by mechanical trauma which bruised tissue and caused extravasation of plasma in the normal connective tissue. The precipitation of acid mucopolysaccharides from the ground substance by substances derived from necrosis of tissue is generally believed to be the mechanism of the formation of fibrinoid.

Phemister[20, 21] believed that freshening of the bone ends was unnecessary for bone grafting in most cases of delayed union; and if an adequate calcific surcharge be placed around the fracture site outside this fibrous tissue and fibrinoid reaction, union would ensue. Some of these cases would no doubt unite with a further 6 to 9 months of immobilization. Therefore, bone grafting is only a part of the process which produces union of a fracture. The so-called shingling operation

will produce union in many cases. Most of these operative procedures act as an adjunct to healing primarily by producing a local increased vascularity. The new bone or periosteal disturbance is merely an assist to the prime factor of increased local vascularity. Re-establishment of this vascular tree by modern blood vessel surgery promises to be a real and outstanding contribution in the maintaining and establishing of bone continuity following fracture.

MODERN TECHNICS OF TREATMENT

For a more practical understanding of fracture healing let us review some of the specific bones of the body and the modern concept of treatment to effect healing and prevent delayed, or nonunion. Remember that restoration of function is more important than a pretty roentgenographic picture.

Upper Extremity

Fingers and Metacarpals: Fingers and metacarpals demand accurate and immediate reduction with adequate immobilization in plaster for approximately 3 weeks with a maximum of 4 weeks. All fingers and joints not involved in a fracture should be kept mobile in so far as possible. Internal fixation should be limited, and used only when manipulative reduction is or would be unsuccessful. Traction to the fingers is an adjunct but is often more dangerous than useful. A good axiom to remember is,—do not overtreat fractures of the fingers and metacarpals, for it is better to maintain motion in the joints than to secure union of the shafts if both cannot be attained. In treatment of nonunion of the phalanges and metacarpals the most practical type of fixation is intramedullary cortical bone grafting followed by simple splinting and rehabilitation (fig. 2).

Carpus: The key to adequate treatment of fractures of the carpal navicular depends on early diagnosis and adequate immobilization for a sufficient period of time to allow solid bony union. This immobilization may be continued for 12 to 18 months provided the metacarpophalangeal joints of the fingers are kept mobile and the wrist is put in no extremes of position. The hand should be maintained in a grasping pose as if one were holding a big orange at the time the plaster is applied (fig. 3). Internal fixation, bone grafting, excision and drilling procedures for fracture of the navicula should be reserved for the unusual and exceptional cases.

Both Bones of Forearm: Treatment of both bones of the forearm remains one of our outstanding problems in fracture therapy. In dealing with children it is advisable that proper alignment always be attained. Length and position are desirable but not essential. In fractures of the proximal third, the forearm should be immobilized in supination, of the middle third, neutral or midposition, and of the distal third in pronation.

These fractures in adults[14] are much more difficult to treat. If there is displacement at one or both fractures, then accurate reduction and maintaining of position is essential. This, as a rule, demands open reduction, adequate internal fixation and bone graft supplements. Probably the best form of treatment is an intramedullary pin in the ulna, a slotted or Eggers[11] type plate on the radius and

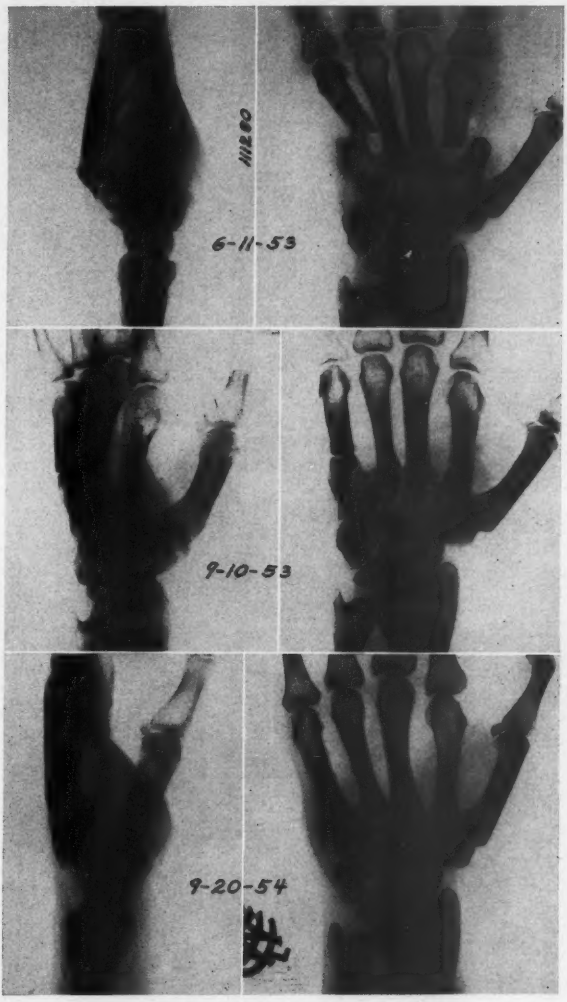

FIG. 2. Shows roentgenograms of the hand of a 24 year old man 5 months after a crushing injury. There was nonunion of second, third and fifth metacarpals. Intramedullary bone pegs were used to restore continuity. Note the grafts have completely lost their identity and the fractures have united in anatomic alignment.

iliac sliver bone grafts placed at both fracture sites. The use of onlay cortical bone grafts is still a well established and acceptable procedure (fig. 4). In cases with nonunion, bone grafting by any of these methods is acceptable.

Elbow: Fractures about the elbow in children[1, 3] demand an accurate anatomic reduction and good fixation to prevent displacement or any incongruity. This

FIG. 3. Shows type of plaster cast used for immobilization of the carpal navicular. Note "grasping pose" and a range of motion of the metacarpophalangeal joint.

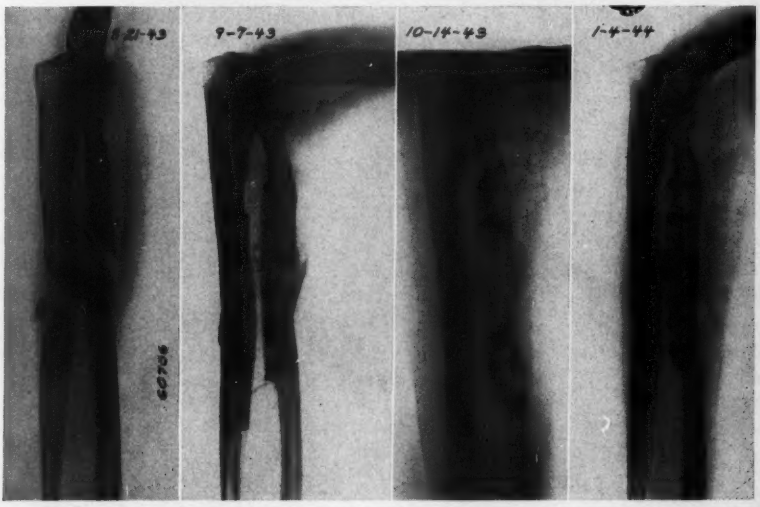

FIG. 4. Showing a fracture of both bones of the forearm at the junction of the middle and proximal thirds which were treated by onlay cortical bone grafts, resulting in rapid and solid union in anatomic alignment and position. At 2 months external fixation was discontinued and active rehabilitation instituted.

general axiom may be applied to practically all joints in children. In adults fractures about the elbow should be reduced as accurately as possible and internal fixation used if necessary to maintain accurate repositioning. However, in the badly comminuted, mangled elbow fractures the British advocate early active exercise at 2 to 3 weeks. They recommend letting motion occur where it will, be it in the joint or at the fracture site. It is essential to remember in treatment and

rehabilitation of fractures about the elbow[26], that passive stretching or manipulation force to restore motion should *never* be employed.

Humerus: Fracture of the humerus[24] for many years was one of the leading contenders for the production of nonunion in long bones. The hanging cast has become the treatment of choice in transverse, oblique, spiral or comminuted fractures of the shaft or surgical neck, and has reduced the incidence of nonunion. It is essential that the 10 following rules for use of the hanging cast be followed:

1. The plaster must be light and never a distracting force.
2. The elbow should be held at 90 degrees, with the plaster extending from the midpalm to the fracture level or not more than 1 inch above.
3. The sling must be fixed at the level of the wrist.
4. The loop for the sling, placed on the dorsum of the wrist, corrects lateral angulation; placed over the volar side, it corrects medial angulation of the fracture.
5. A long sling corrects posterior, a short one, anterior angulation.
6. The arm must be continously dependent, i.e., in traction.
7. Anteroposterior and lateral roentgenograms should be made at weekly intervals or more often if indicated.
8. Early, active, vigorous exercise of the "*longitudinal muscles*" of the shoulder, 4 to 6 times daily, is imperative.

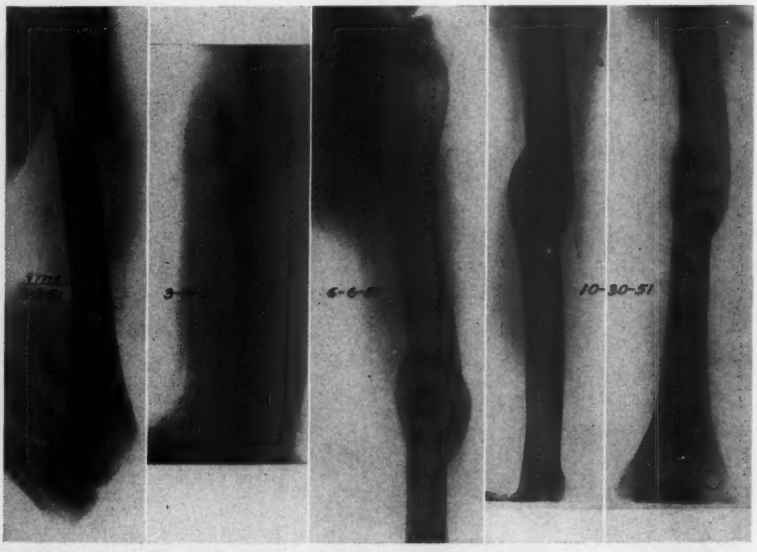

FIG. 5. Shows a transverse fracture in the humerus of a 35 year old woman which was treated with a hanging cast. At the end of 1 month there was abundant callus formation but the fracture line was widened not only of the parent bone but through the ball of callus. In spite of this a hanging cast was continued and in another 3 weeks the fracture site had tightened allowing no clinical motion. At the end of 9 months from the time of fracture the remodeling of the humerus was essentially complete and the patient was asymptomatic. This patient returned to active duty as a public health nurse at the end of 2 months from the date of the fracture.

9. Systematic resistant exercises of the fingers and thumb are essential.

10. If the patient must remain recumbent, 3 to 5 pounds of traction should be maintained at the elbow in line with the humerus.

There is a rich vascular bed and good musculature about the humerus, and with this adequate blood supply union will proceed rapidly in the presence of motion. Often when nonunion appears inevitable, rigid immobilization is not necessary if your patient is cooperative (fig. 5).

Fractures involving the capital part of the humerus where there is comminution of a "*split head*" type, accurate repositioning and fixation by operative methods is mandatory if good function is to be attained (fig. 6). In the extremely comminuted humeral heads, replacement prosthesis has proved of some advantage but still has a very limited field. The Lawrence Jones resection operation with reattachment of the rotator cuff to the shaft of the humerus has proved of benefit in some cases where reassembly of the humeral head is not possible. If the head can be repositioned so that the articular portion faces the glenoid, incongruity and deformity may persist in the remaining portion of the head but the functional result will be good (fig. 7).

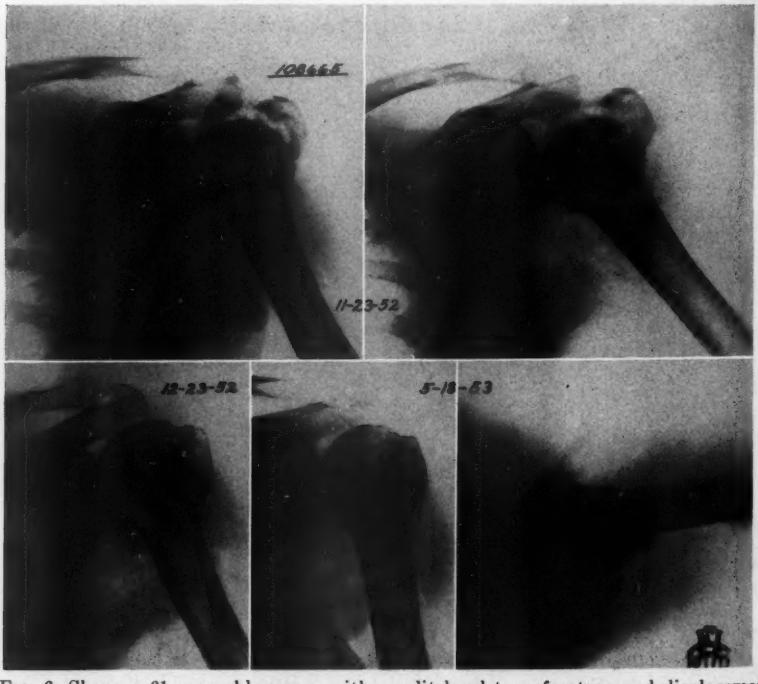

FIG. 6. Shows a 61 year old woman with a split head type fracture and displacement of the greater tubercle. Open reduction, internal fixation with one screw for the split head and two wire loops for the trochanteric rotator cuff detachment resulted in an excellent result. Postoperative immobilization was on an abduction humerus splint for 3 weeks, then active exercise was begun. At the end of 6 months the patient had full normal range of motion in all directions with excellent function of the shoulder with no pain.

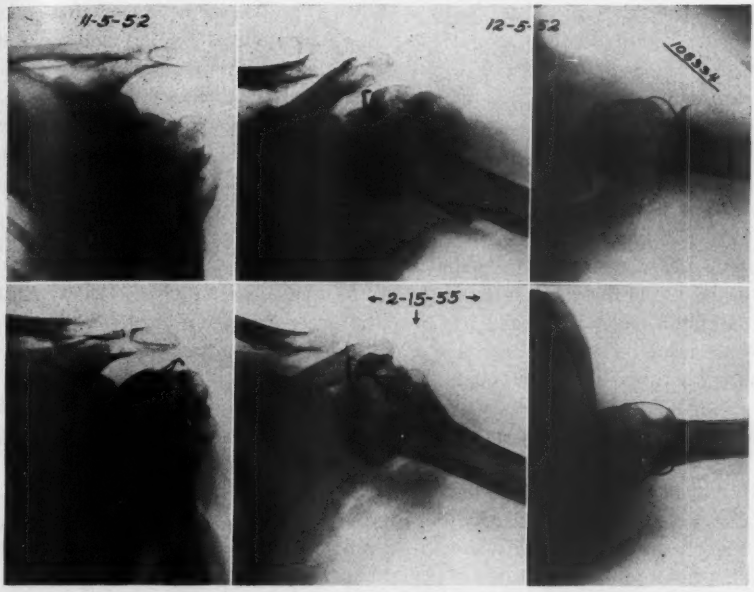

FIG. 7. Shows a very badly comminuted displaced fracture of the head of the humerus in a 60 year old woman. By open reduction and internal fixation with multiple wire and catgut sutures the head was reassembled so that there was a large articular surface facing the glenoid. Immobilization on an abduction humerus splint was continued for 5 weeks, then rehabilitation instituted. Twenty-six months postoperative the patient has an excellent range of motion, 95 per cent of normal in all directions. She has no pain and an excellent result.

Clavicle: It is difficult to produce nonunion in a fracture of the clavicle. In the majority of cases a figure of eight strapping made of stockinette or cotton flannel bandage reinforced with adhesive tape and strengthened every 3 to 5 days will be adequate. The maintaining of length and alignment will result in adequate union and good function. If there is a bony prominence which persists for more than 12 to 18 months, postinjury, and is cosmetically unsightly, it can be removed surgically. It is rarely necessary to resort to operative reduction and internal fixation for fracture of the clavicle.

LOWER EXTREMITY

The Foot: In treatment of fractures of the toes and metatarsals manipulative reduction and immobilization in plaster is adequate in the majority of instances. However, if there is gross displacement of the metatarsal heads and they cannot be repositioned by manipulation, then open reduction and intramedullary fixation is indicated. Pins should be passed through the plantar surface of the foot through the head of the metatarsal and down the medullary canal for fixation.

In simple fractures of the neck or head of the talus, reduction and prolonged protection from weightbearing is the procedure of choice. In fractures of the

talus with displacement, aseptic necrosis of the body may be anticipated and an early subtalar fusion or triple arthrodesis is often advisable. In fractures of the os calcis the simple fractures without subtalar involvement are easily treated by immobilization in plaster. Those with depression of the articular facets or in cases of extensive subtalar involvement respond best to early triple arthrodesis with a reduction of the lateral spread of the body. Immobilization and nonweight-bearing must continue for 8 to 12 weeks, followed by an equal period of constructive rehabilitation.

The Ankle: Fractures about the ankle, particularly the Potts and Cottons fractures, demand accurate reduction and in most instances internal fixation, at least, of the medial malleolus. Since the ankle bears more weight than any joint of the body, prolonged protection is necessary. Motion may be begun at 6 to 8 weeks, but weight-bearing should be deferred as a rule for 3 months, in all major fractures about the ankle.

In incongruous fractures with displacement and malunion or nonunion the general rule is fusion of the ankle, as the procedure of choice. We have found that this is best accomplished by the contact compression arthrodesis methods as advocated by Key[13] and popularized by Charnley[9].

Lower Leg: In dealing with fractures of both bones of the lower leg one can disregard the fibula unless it involves the distal 3 inches. Practically all authorities on treatment of fractures of the tibia agree that manipulative reduction and plaster immobilization is the procedure of choice. The oblique and spiral fractures of the tibia with fractures of the fibula are very prone to slip even though an initial anatomic reduction is obtained. Absorption at the fracture site occurs between the second and third weeks and slipping results producing a malposition. The procedure of choice in these oblique fractures is open reduction and fixation with one or more screws or with a parallel wire loop, that is, parallel to the fracture line. In rare instances a slotted Eggers type plate may be advisable or an intramedullary nail as advocated by Lottes[15] (fig. 8).

For established nonunion in fractures of the tibia several acceptable procedures are available: they are, (1) onlay cortical bone graft of autogenous or homogenous bone placed on the lateral side of the tibia, (2) iliac bone placed around the fracture site according to Phemister's technic, (3) osteotomy of the fibula with plaster immobilization and early ambulation, (4) in large defects, which must be bridged to maintain length, dual onlay bone graft as advocated by Boyd[4] is an acceptable procedure, (5) the hemicylindrical graft as advocated by Flanagan[12], may be employed as a last resort, and (6) the resection and leg shortening as advocated by Urist may be used in preference to amputation.

The Knee: Fractures into the knee joint involving the tibial plateau or the femoral condyles must have anatomic reduction with adequate fixation. If this reduction can be attained and maintained by external fixation then this is the procedure of choice. If not, open reduction and fixation with stainless steel screws or pins becomes necessary (fig. 9). Motion can be begun in the joint between 6 and 8 weeks but weight-bearing should not be allowed until the structures are radiographically solid, which, as a rule, require a minimum of 3 months.

The Femur: In the past 15 years the treatment of fractures of the femur has

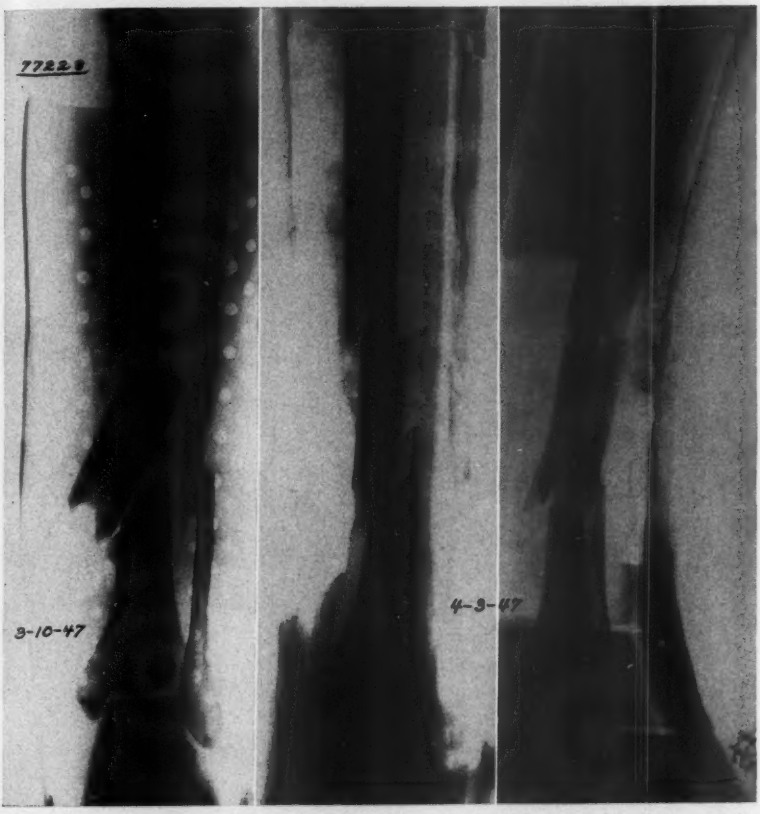

FIG. 8. Shows a segmental comminuted fracture of the tibia treated by intermedullary fixation with a Lottes type nail.

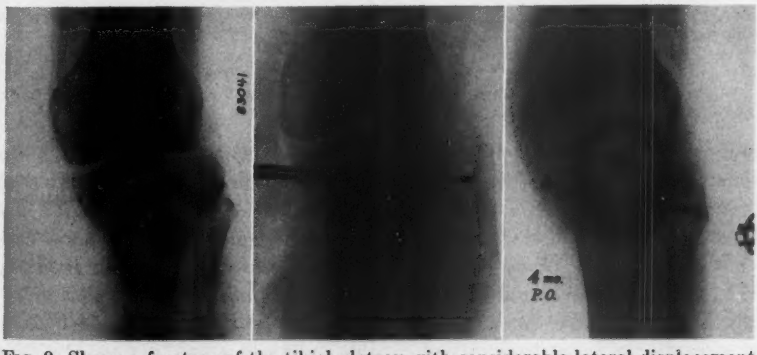

FIG. 9. Shows a fracture of the tibial plateau with considerable lateral displacement of the lateral tibial condyle which has been reduced and maintained in position by two Knowles pins. Four months postoperative the fracture is healed and the fracture line is practically invisible.

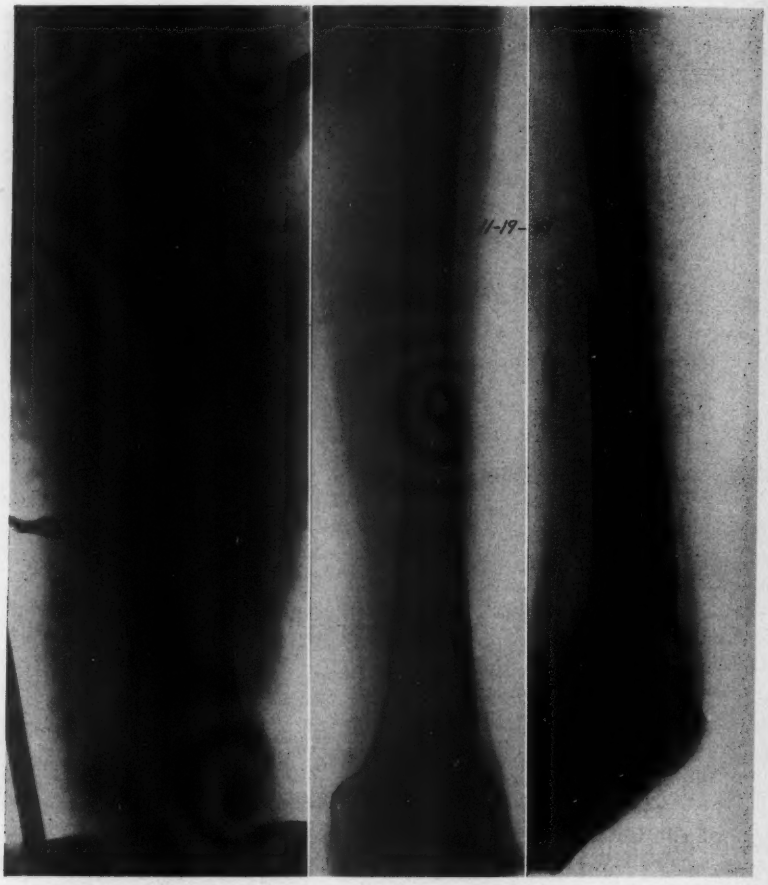

FIG. 10. Shows a transverse fracture in the distal portion of the middle third of the femur which has been fixed by open reduction and internal fixation with a Kuntscher type of intramedullary nail. Weight-bearing was allowed at the end of 1 month; no external fixation was utilized at any time. Patient was back at full activity at the end of 3 months. Follow-up roentgenograms made at 7 months after injury shows solid bony union in proper alignment and position with the nail remaining in place.

made great progress. For many years the accepted treatment of fracture of the femur was rigid immobilization in a body plaster spica. Russell's traction gave adequate results in a substantial number of cases. At the beginning of World War II the practice of skeletal traction with a Thomas ring splint and Pearson attachment was the treatment of choice. The traction maintained good alignment, length, and as a rule good position but did not force rigid fixation. Since there is an abundant blood supply to the femur we know that rigid fixation is not essential, and that early active exercise enhances union. Active exercise not

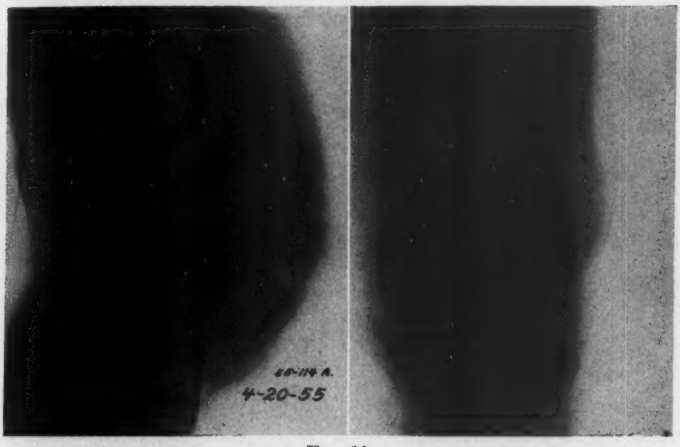

FIG 11a

FIG. 11. Shows a patient with a supracondylar fracture of the femur treated by traction with a pin in the proximal tibia and distal femoral fragment. Alignment and position are easily restored and union is rapid.

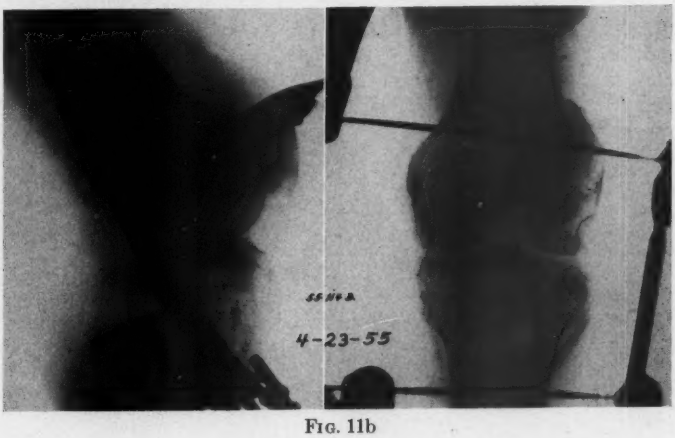

FIG. 11b

only increases the active circulation but there is a suggestion that the end product of muscle combustion may act as a catalyst to bone production.

Since World War II the perfecting of intramedullary[22] fixation of fractures of the proximal two thirds of the femoral shaft has been established as the procedure of choice (fig. 10). With intramedullary fixation, weight-bearing can be allowed early, in 2 to 4 weeks, and the muscle turgor and strength are maintained. Joint function remains normal and prolonged hospitalization and immobilization are eliminated. However, in the badly comminuted fractures involving the

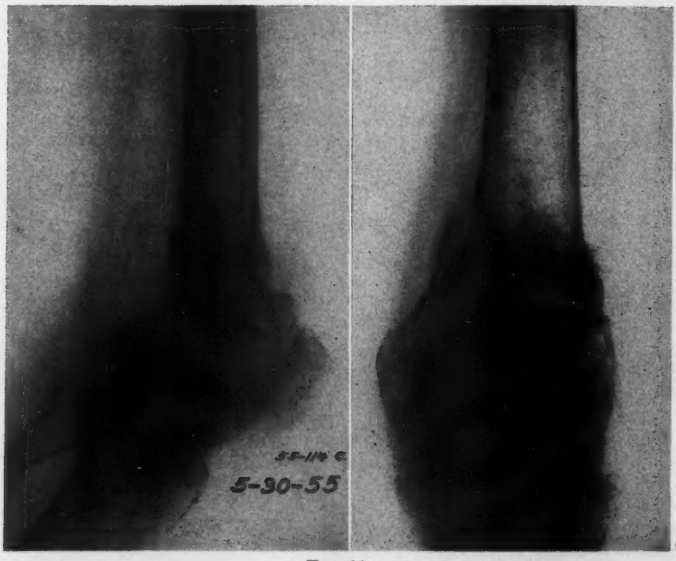

FIG. 11c

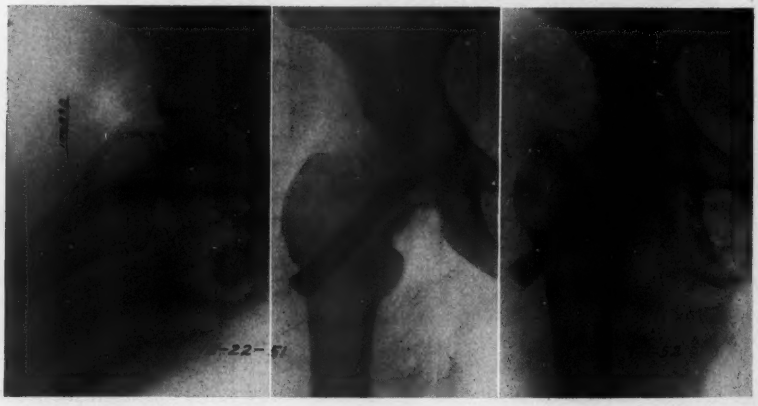

FIG. 12a

FIG. 12. Shows a fracture of the neck of the femur which had healed in a 42 year old woman. Two years later she developed a pathologic fracture through the base of the capital portion of the femur with marked sclerosis and vascular changes of the head. Osteotomy and bone graft resulted in rapid healing of the pathologic fracture and excellent progress in revascularization of the head. Joint space was maintained. Patient is now walking without pain or external support of any type in performing daily activities as a receptionist in a large restaurant.

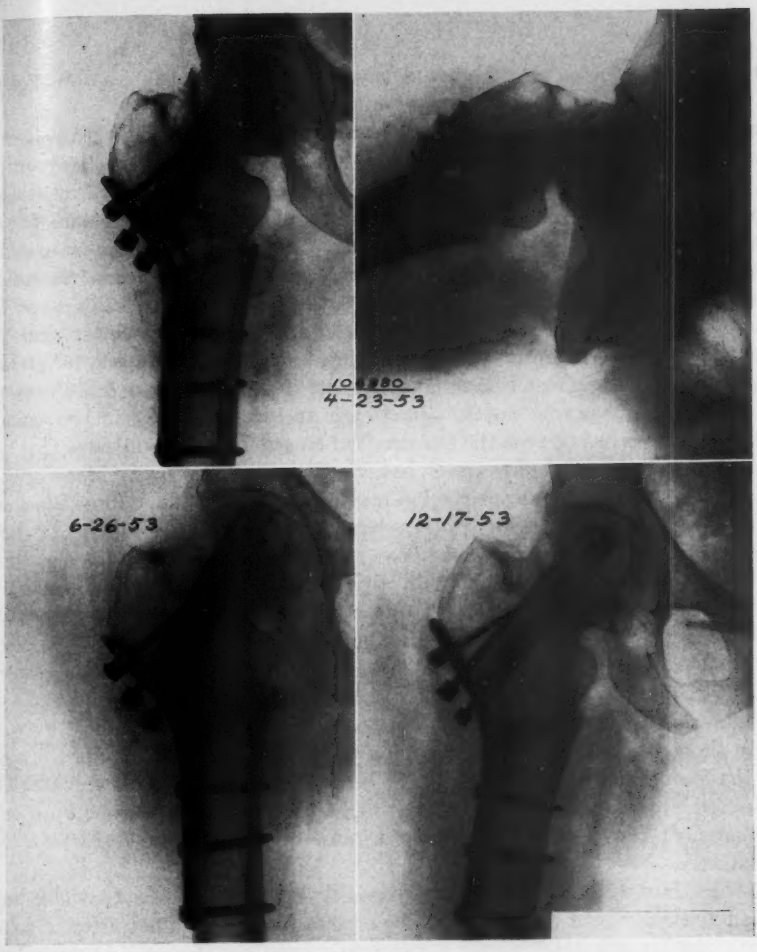

FIG. 12b

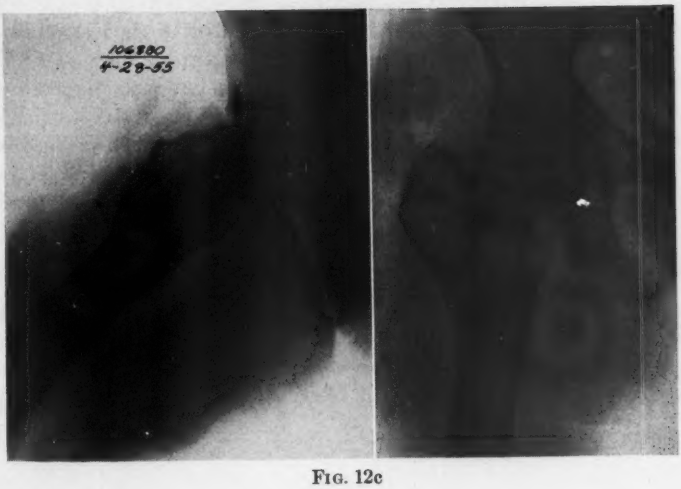

FIG. 12c

femoral shaft or in fractures of the lower third of the femur the treatment of choice remains two pin skeletal traction in the Hodgen type splint (fig. 11).

In established nonunion of fracture of the femur, intramedullary fixation and iliac bone grafting or onlay cortical grafting are the procedures of choice.

Fractures of the Femoral Neck: The body plaster immobilization as used 25 years ago for fractures of the neck and intertrochanteric fractures has fortunately been replaced by internal fixation. For the intertrochanteric fracture of the femur various forms of angle nails have proved adequate. The Jewett nail, the Neufeld nail, the Thornton or McLaughlin attachment to the Smith-Petersen nail and other similar nails give good support if properly inserted. In patients over 70 years of age who sustain a fracture of the neck of the femur the replacement prosthesis has become the treatment of choice for many surgeons.

This so-called "*unsolved fracture*" has run the gauntlet of many procedures in the past 25 years. The reconstruction operations of Whitman, Albee, Brackett, Magnuson, Colonna, and many others have been used in treating this fracture. Various forms of osteotomies have been advocated, tried, and many discarded. Most of these osteotomies take their origin from the old Von Bauer, Lorenz or Schanz procedures. At the present time the two most accepted types are the McMurray high displacement, and the Blount[2] lower angulation osteotomy with internal fixation by Blount as modified by Moe. In nonunions of the femoral neck with an obviously viable head, the Blount osteotomy has given satisfactory results when done with precision in the hands of competent surgeons.

In those cases of established nonunion with obvious avascularity of the head of the femur, neither a McMurray nor Blount operation will be adequate. The accepted procedure in most of these cases is a reconstruction procedure or a replacement prosthesis with the stem or more often an intramedullary type of prosthesis.

In the cases with nonunion which range between the extremes of a viable head or an avascular head there seems to be a place for another innovation in treatment; that is osteotomy combined with bone grafting. In 1947 Dickson[10] advocated his geometric osteotomy with iliac bone grafting through a window in the neck of the femur. He reported a substantial number of good results by this method. An angulation osteotomy with cortical bone grafts may offer a better, and less shocking procedure. These grafts pass from the osteotomy site across the neck of the femur into the head for the purpose of accelerating union of the neck and establishing a new channel of blood into the head to prevent further avascular necrosis. In a small series of 16 of our cases this procedure has appeared most encouraging. Figure 12 illustrates this type of osteotomy and bone graft.

SUMMARY

The outstanding features of the progress in the treatment of fractures in the past 25 years depend upon developments in many fields. As far as fractures per se are concerned the emphasis has been increasingly placed upon the problem of maintaining or establishing adequacy of blood supply to the area of involvement.

The most outstanding single contribution has been the development of modern antibiotic therapy. Of vast importance are the advances in metallurgy and anesthesiology. The establishment of blood banks and bone banks have made outstanding contributions in the treatment of fractures.

Intramedullary fixation and advances in bone grafting have been great adjuncts to the armamentarium of the fracture surgeon.

Plastic and vascular surgery are making outstanding contributions to the restoration of the local blood supply and prevention of nonunion.

Modern rehabilitation of the musculature and joints has given not only physical but great emotional and economic assistance in the preservation and restoration of function.

869 Madison Ave.
Memphis 3, Tenn.

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PSOAS MYOSITIS OR FIBROSITIS IN THE DIFFERENTIAL DIAGNOSIS OF LOWER ABDOMINAL DISEASE

WILLIAM M. BLAIR, M.D.

Wharton, Texas

The diagnosis and treatment of lower abdominal pain is an ever recurring problem which constantly tests the diagnostic acumen and surgical judgment of the surgeon. Over the years our thinking has been directed toward pathologic lesions of the viscera in such cases. Abnormalities of the tissues of the abdominal wall, anteriorly and posteriorly, are often not considered.

The syndrome of psoas myositis or fibrositis was described by Greene[4] in 1945. This syndrome came to my attention in 1946 when Dr. Greene was asked to see a patient in consultation, who had lower abdominal pain. All clinical, laboratory, and radiologic studies were within normal limits. The patient was much concerned about his condition and asked that an exploratory laparotomy be done. A diagnosis of psoas myositis was made and the pain subsided on salicylate therapy. This stimulated my interest in the syndrome. Several other cases, in which the patient had lower abdominal pain were diagnosed as psoas myositis. There was a dramatic relief of the symptoms when these patients were given salicylates.

Fibrositis has been used to designate pains and aches of musculoskeletal origin since the introduction of the term by Gowers in 1904[3]. The term "*myositis*" denotes a reactive process in muscle of a type usually related to the presence of infection. Myalgia and myositis frequently are used as synonyms of fibrositis. A true definition of fibrositis based on pathophysiology has not been made. Gutstein[5, 6] has suggested the term *myodysneuria*. Myodysneuria denotes localized functional sensory and/or motor abnormality of musculoskeletal tissues. It is the purpose of this paper to call attention to diseases related to the psoas muscle and the problems of differential diagnosis of lower abdominal pain rather than to discuss the various types of musculoskeletal disease.

ETIOLOGY

The etiology of this syndrome is never clear cut. In the majority of patients, nervous tension is an important factor. Trauma, defects in body posture, or acute infection may be the exciting cause. It very frequently follows pregnancy. In such cases, the mother, after having had a trying prenatal course, finds that there are not enough hours in the day for rest. Hence fatigue, physical and mental with irritability and depression follow.

DIAGNOSIS

Psoas muscle syndrome is often mistaken for appendicitis, salpingitis, cystic ovary, colitis, and the all inclusive chronic pelvic inflammatory disease. The syndrome is more frequent in women than in men.

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The majority of patients of psoas syndrome fall into the hyposthenic type—tall, thin, poorly developed musculature, and the asthenic type with a wide roomy pelvis and sagging of the abdominal viscera. They usually are underweight with poor posture and poor gait. Not all are underweight, however, The asthenic type may be overweight. The personality pattern in these patients is very similar. They are tense, apprehensive, and are extremely anxious regarding their condition. Depression, anxiety and irritability are prominent. Approximately 50 per cent of the patients who are examined have either had some type of abdominal surgery, with no relief, or have been advised that they need an operation. Some have been labeled psychoneurosis.

The subjective manifestation of the psoas syndrome is pain in the lower quadrants of the abdomen. It may be unilateral or bilateral. It occurs more frequently on the right than on the left side. The pain is described as dull and aching in character. It is persistent, lasting for days, weeks, months, and sometimes years. Acute exacerbations occur. The pain is aggravated by activity such as walking, climbing, stooping, bending, lifting or any activity that increases the use of the iliopsoas muscles. The pain diminishes with rest. Every patient will state that pain is improved by lying down and is always aggravated with activity. The onset of pain may be sudden and acute or it may be insidious in its onset. Exacerbations and remissions of the symptoms are common. However, the pain may persist for long periods. Sacroiliac joint pain and pain in other muscles frequently are associated with pain in the psoas muscle.

The only definite objective finding is tenderness over the psoas muscle. Frequently rigidity and muscle spasm can be felt. The muscle is usually tender from its origin to its insertion. At times it will feel almost board-like in character. In examining for psoas muscle tenderness, an attempt is made to put the patient at ease, so that the abdominal muscles will be as relaxed as possible. If other diseases are present, this is most difficult. Appendicitis, caecal disease and other conditions cause spasticity of the muscles of the abdominal wall and deep palpation becomes impossible.

In the routine examination of women patients, a vaginal examination is made first. The patient is always catheterized, on the examining table, before vaginal examination. The condition of the pelvic viscera is determined before any attempt is made to palpate the psoas muscle. Informing the patient that there is no abnormality in the pelvis is reassuring and helps the patient to relax and makes the remainder of the examination easier. After the pelvic examination is made, the abdomen is palpated. Gentle pressure is applied, first in the right lower quadrant, and the colon is displaced medially, when possible, allowing the psoas to be palpated both in the upper and lower abdomen. Deep palpation with gentle pressure causes the patient to complain of pain. Increasing pressure demonstrates the psoas muscle and the psoas muscle tenderness. The leg is then flexed at the hip, with the knee extended, and the examiner elevates the leg without the voluntary aid of the patient. One hand is used to lift the leg while the other hand palpates the psoas muscle. The patient will have pain on palpation of the muscle. Then with the examining hand still on the psoas muscle, the other hand is removed from supporting the leg, and the voluntary muscles are brought into

TABLE I

Tabulations of nine patients with diagnosis of psoas myositis

1. S. B.	F-W-M-34	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—appendix normal; psoas muscle—fibrous myositis
2. R. T.	F-W-M-24	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—fibrosis of adjacent adipose tissue; adenomyoma of fallopian tube
3. V. T.	F-C-S-19	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—appendix normal; psoas muscle—myositis
4. K. K.	F-W-S-15	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—psoas myositis
5. J. B.	F-W-M-22	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—psoas muscle—focal hyaline changes; fallopian tubes—normal
10. H. B.	F-W-M-36	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—psoas muscle normal; appendix—normal
17. S. L.	M-W-M-45	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—psoas muscle normal; appendix normal
18. C. R.	F-LA-M-26	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—appendix normal; psoas muscle—focal degenerative changes
20. L. K.	F-W-S-20	Preoperative diagnosis—psoas myositis. Pathologic diagnosis—appendix pinworms present; psoas muscle—normal

Note. In this table of the 9 patients thought to have psoas myositis preoperatively, histologic changes were demonstrated in the psoas muscle in 5 and in the adjacent fat in 1 The histologic changes are shown in figure 1.

play. The patient attempts to hold the leg up, the psoas muscle contracts, and the pain is much intensified, sometimes causing outcry because of excruciating pain.

The laboratory findings in these patients usually are within normal limits. There may be slight temperature elevation from 99.6 to 100.2 F. This usually occurs when several other muscles are involved.

BIOPSY STUDIES

The question arose as to whether or not histologic confirmation of the diagnosis could be established. The diagnosis of this syndrome in Greene's[4] cases was based upon clinical findings. Biopsy of the psoas muscles had not been done. It was decided to biopsy the psoas muscle in patients diagnosed preoperatively as having psoas myositis, in patients with acute inflammation of surrounding viscera, and in patients with noninflammatory visceral disease, to determine if histologic changes were present in the muscle. In 9 patients thought to have psoas myositis, an acute surgical condition could not be excluded. Five of these patients had histologic changes in the psoas muscle, and 1 in the adjacent fat (table I, fig. 1). After abnormalities were demonstrated in cases of psoas myositis, biopsies were obtained from the psoas muscle in 5 additional patients with acute inflammatory diseases of the adjacent viscera, to determine whether or

FIG. 1. Voluntary muscle with adjacent increased cellularity involving adipose tissue. Nuclei resemble sarcolemma nuclei or young connective tissue cells.

TABLE II

Acute inflammatory disease in viscera with no change in psoas muscle

Five patients with acute inflammatory disease in viscera (acute appendicitis) with no change in psoas muscle.

9. S. M.	F-W-M-23	Preoperative diagnosis—appendicitis. Pathologic diagnosis—appendicitis, acute
11. A. B.	F-LA-S-16	Preoperative diagnosis—appendicitis. Pathologic diagnosis—appendicitis, subacute
13. A. R.	F-W-S-20	Preoperative diagnosis—appendicitis. Pathologic diagnosis—appendicitis, acute
14. O. Y.	F-LA-S-16	Preoperative diagnosis—appendicitis. Pathologic diagnosis—appendicitis, acute
16. E. W.	M-C-S-12	Preoperative diagnosis—appendiceal abscess. Pathologic diagnosis—appendicitis, acute, suppurative

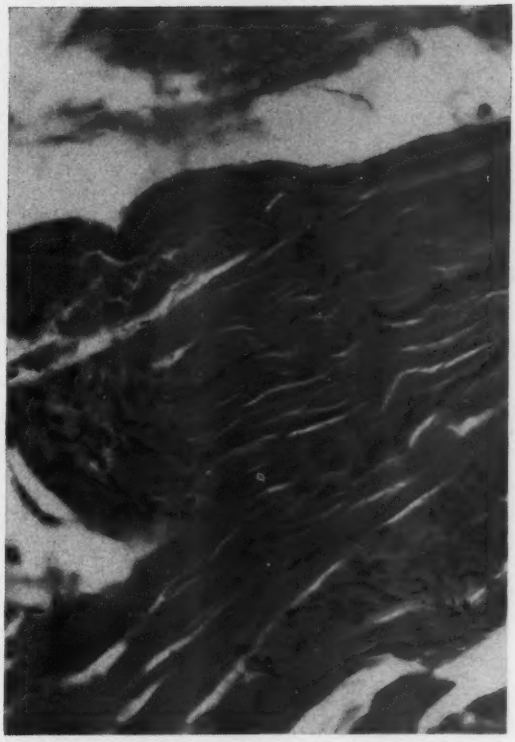

FIG. 2. Voluntary muscle with focal increase of nuclei of sarcolemma type

not the adjacent inflammatory disease produced changes in the muscle. These patients did not show any pathology in the psoas muscle (table II). In addition, biopsies were obtained in 9 patients with noninflammatory disease, to determine whether or not histologic changes were present in the muscle of patients unsuspected of psoas disease. In this group evidence of disease was not found in the psoas muscle (table III).

DISCUSSION

The skeletal muscle is a complicated structure; it contains, in addition to connective tissue, blood vessels, lymphatics, and nerves, muscle fibers or *myones* which constitute the histologic units. In the body, muscle fibers are excited only by impulses arriving over motor nerves.[2]

Musculoskeletal diseases are divided into (1) congenital defects of skeletal muscle, (2) muscular dystrophies, (3) inflammatory diseases of muscle (named according to type of infection) as suppurative, viral, syphilitic, tuberculous, parasitic, and fungus, (4) tumors, (5) traumatic and circulatory diseases, (6) neuromuscular atrophies, (7) toxic and metabolic diseases, and (8) disorders of

FIG. 3. Foreign body giant cell and increase in sarcolemma cells

TABLE III

Eight patients with noninflammatory visceral disease who had biopsy study of the psoas muscle

6. L. M.	F-LA-M-20	Operation—salpingectomy for sterilization. Pathologic diagnosis—psoas muscle normal; appendix—normal
7. I. H.	F-C-M-23	Operation—appendectomy. Pathologic diagnosis—appendix normal; psoas muscle—normal
8. O. M.	F-W-M-25	Operation—appendectomy. Recurrent appendicitis. Pathologic diagnosis—appendicitis, obliterative; psoas muscle—normal
12. A. S.	F-W-M-16	Operation—elective appendectomy. Pathologic diagnosis—appendix normal; psoas muscle normal
15. M. B.	F-W-S-16	Operation—appendectomy. Pathologic diagnosis—appendix normal; psoas muscle normal
19. W. H.	M-W-S-19	Operation—appendectomy. Pathologic diagnosis—appendix normal; psoas muscle normal
21. E. L.	F-LA-M-27	Operation—appendectomy; salpingectomy. Preoperative diagnosis—pelvic inflammatory disease. Pathologic diagnosis—fallopian tube normal; psoas muscle normal
22. V. C.	F-C-M-24	Operation—appendectomy. Pathologic diagnosis—appendix normal; psoas muscle—normal

excitability and contractability.[1] Further mention of these conditions is not within the scope of this paper.

Pain can be provoked by injury of muscle, or the aggravated stimulation of muscle, bone and periosteum, joints, tendons and fascia, arteries, and the thoracic or abdominal viscera. Muscles, tendons and fasciae are especially susceptible to painful stimulation by chemical agents. Muscle is relatively insensitive to pricking or cutting, but pain is aroused by pressure, pinching or squeezing, or by exercising under ischemic conditions. Tension acts, also, as a pain stimulus for muscle, tendon or fascia. Deep pain is diffuse, usually continuous and poorly localized. Pain occurring in ischemic muscles during activity is due to chemical irritants produced by the active tissues which accumulate and stimulate the nerve endings when the circulation to the part is arrested or considerably reduced. The soreness of healthy muscles which comes on some hours after exercise is of the same nature.[7]

SUMMARY AND CONCLUSIONS

The psoas fibrositis or myositis syndrome is discussed and the method used in examining for tenderness of the psoas muscle is explained.

Twenty-two patients from whom muscle biopsies have been taken, are tabulated. Six of these patients have shown some type of psoas muscle change on pathologic examination. Five patients with acute appendicitis did not show any change in the psoas muscle. This was also true in some patients with noninflammatory disease of the abdominal viscera.

A positive statement that the changes in the psoas muscle were responsible for the pain cannot be made. However, the pathologic findings of change in the muscle definitely indicate that lower quadrant pain may be caused by other conditions than disease of the intrapelvic or intra-abdominal viscera. Enthusiasm for the diagnosis of psoas fibrositis has been tempered with caution. If there was some question of disease of the intra-abdominal or intrapelvic viscera, patients were operated upon.

I wish to express my appreciation to Dr. Stuart A. Wallace, professor of pathology, Baylor University College of Medicine, Houston, Texas, for the pathologic reports and to Dr. James A. Greene, professor of medicine, Baylor University College of Medicine, Houston, Texas, for his assistance.

210 N. Houston
Wharton, Texas

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ACUTE PANCREATITIS WITH NECROSIS AND PERFORATION OF COMMON BILE DUCT: CASE REPORT

DONALD B. BUTLER, M.D., RALPH EICHHORN, M.D.

Houston, Texas

The complications of acute pancreatitis are manifold. Cardiovascular collapse, secondary infection with spreading peritonitis, and massive hemorrhage are some of the more severe catastrophes which may occur following hemorrhagic pancreatitis. It is surprising that the common duct, with its close anatomic relation to the head of the pancreas, is not more often the site of injury during attacks of acute hemorrhagic pancreatitis. As the head of the pancreas becomes involved in an acute necrotizing hemorrhagic process the entire pancreatic portion of the common bile duct may, on occasion, become compressed by edema producing transient jaundice. But, in general, permanent damage to the common bile duct is a rare sequela of acute pancreatitis.

Zaslow[1] in 1953, described the cases of 2 patients in whom acute pancreatitis resulted in perforation of the common bile duct. A review of the voluminous literature on complications and sequela of acute pancreatitis fails to reveal any other reports of clinical cases of pancreatitis associated with necrosis and perforation of the common bile duct.

In 1953, the authors had an opportunity to observe and treat a patient with acute pancreatitis in whom perforation of the common bile duct with bile peritonitis was observed and successful treatment was instituted. A review of the findings and experiences in this case form the basis of this report.

CASE PRESENTATION

A. G., 38 year old white woman, was admitted to Hermann Hospital on Sept. 16, 1953. with the complaint of right upper quadrant pain, nausea, and vomiting, and chills and fever of three days duration. The pain was constant and severe with intermittent exacerbations. There was no radiation of pain. For 26 years she had had recurrent mild attacks of mild right upper quadrant nonradiating pain, nausea, and vomiting. She had noted intolerance to fatty and greasy foods. One month prior to the present illness, the patient had a gallbladder roentgenologic examination which revealed a nonfunctioning gallbladder.

On physical examination an acutely ill, toxic white woman was seen who was moderately obese and had a temperature of 101 F., pulse 96 per minute, respiratory rate 18 per minute, blood pressure 140/70. The patient appeared quite dehydrated and seriously ill. The right upper quadrant was exquisitely tender with marked rigidity. The gallbladder was palpable and quite tender. There was moderate abdominal distension. No jaundice could be detected, clinically. The rest of the physical examination was essentially negative including pelvic and rectal examination. Laboratory examination revealed a hemoglobin of 14.2 mg. per cent, erythrocyte count 4,910,000 per cu. mm., leukocyte count of 23,850 per cu. mm. with a left shift. Serum amylase was reported as 95 mg. per cent (normal 20 to 40 mg. per cent), serum lipase was reported as 0.43 cc. of 1/10 normal sodium hydroxide. Serum potassium was reported 13 mg. per cent, blood sugar 97 mg. per cent, blood urea nitrogen 8 mg. per cent,

From the Departments of Surgery and Medicine, Baylor University College of Medicine, Houston, Texas.

serum chloride 546 mg. per cent, carbon dioxide combining power 50.4 volumes per cent, serum bilirubin 2.9 mg. per cent, urine analysis revealed no acetone and no remarkable microscopic findings.

Roentgenologic examination of the abdomen revealed two or three loops of small intestine in the lower and midabdomen, which were distended with fluid and some gas.

Our clinical impression at that time was acute cholecystitis with stones with pancreatitis —either primary or secondary. The patient was observed carefully for 2 hours and given intravenous fluids and antibiotic therapy, and was taken to surgery for exploratory laparotomy that evening. The abdomen was opened through a right subcostal incision, and the peritoneal cavity contained clear bile. There was extensive fat necrosis throughout the entire abdomen, involving the omentum and mesentery of the small bowel. A large acutely inflamed reddened tense gallbladder was visible adherent to the greater omentum and to the transverse mesocolon. The foramen of Winslow was obliterated with dense adhesions and edema. On opening the foramen, profuse amounts of bile-stained fluid poured from the lesser space. The gallbladder was completely mobilized and the cystic duct was found to be quite small. The region of the common duct was quite edematous and there was marked inflammatory exudate over the entire area. The transverse mesocolon was the site of extensive hemorrhagic fat necrosis, and it was thought inadvisable to enter this area. The common duct was exposed through a shaggy necrotic peritoneum and was found to be dilated to approximately ¾ inch. in diameter. The common duct was opened through a longitudinal incision just distal to the entrance of the cystic duct. Dark bile poured from the opening under moderately increased pressure. On examining the inner surface of the common duct it was seen that there was no posterior wall, but a large cavity extending into the head of the pancreas. This cavity was filled with bile, and after evacuating the bile, much necrotic gray tissue extruded. After exploration of this region, it was apparent that there had been complete necrosis of a portion of the head of the pancreas with sloughing of the medial wall of the common bile duct in its pancreatic portion. The superior mesenteric artery and vein could be palpated and seen within the floor of this large cavity. There was, surprisingly, no excessive bleeding from the area. The proximal end of the common bile duct was readily found, and scoops could be inserted with ease into both the right and left hepatic ducts. The distal end of the common duct could not be readily located due to the intense reaction in the pancreatic region. The duodenum was then opened and the ampulla of Vater was recognized with great difficulty due to the intense inflammatory reaction. A probe was placed into the distal end of the common bile duct and a no. 16 French T tube was threaded through the opening in the common duct into the duodenum. The anterior wall of the common duct was then sutured over the T tube. It was impossible to suture the posterior wall of the common duct of the pancreatic portion as there was no tissue left in which to place sutures. The gallbladder was then examined and the tip was amputated. Thick bile was evacuated from the gallbladder but no stones were seen. The mucosa of the gallbladder was necrotic and sloughed freely within the cavity. A loop of proximal jejunum was mobilized and a cholecystojejunostomy was performed.

Ligation of the common duct proximal to the area of necrosis was strongly considered so that the cholecystojejunostomy would serve as a source of biliary drainage, but the surgeon was hesitant to perform this procedure due to the small diameter of the cystic duct and due to the extreme degree of necrosis and edema within the gallbladder. It was feared that the cholecystojejunostomy would not function for any length of time.

POSTOPERATIVE COURSE

The patient appeared quite toxic, postoperatively, with marked temperature elevation, but there was good bile drainage from the T tube. Serum amylase fell to 32 mg. per cent by the first postoperative day and was within normal limits for over 2 weeks.

On the seventh postoperative day, the patient developed marked mental confusion and exhibited a typical catatonic reaction. It was the impression, at that time, that the patient was suffering from a severe toxic psychosis. On the eighth postoperative day, the psychosis

had subsided. On the ninth postoperative day, the Levine tube was removed from the stomach and within 24 hours it became apparent that the patient had at least a partial pyloric obstruction. The patient was receiving 50 mg. of Banthine every 6 hours plus 20 units of regular insulin every 12 hours. On the seventeenth postoperative day, the patient developed severe epigastric pain radiating to the back and T tube drainage became bloody with clots. The patient also vomited some blood, and blood drained through the drain site in the abdominal wound. At this time prothrombin activity was 35.5 per cent of normal activity. Large doses of intravenous Vitamin K and Vitamin K-1 were given, and fresh blood was given. By the nineteenth postoperative day, the bleeding had stopped, but the patient was still vomiting. On the twenty-first postoperative day, a cholangiogram was obtained which revealed that the distal end of the T tube apparently entered the duodenum through a false passage. The proximal portion of the common duct and the hepatic duct appeared normal, however. On the twenty-second postoperative day, roentgenologic examination of the stomach revealed complete obstruction of the antrum, or pylorus, by extrinsic pressure.

It was our clinical impression that the patient had pyloric obstruction from a large psuedocyst formed in the region of the head of the pancreas. Accordingly, surgery was advised again on the twenty-sixth day. The abdomen was opened through a left rectus incision, and surprisingly, the left upper quadrant was entirely free of adhesions or evidence of fat necrosis. No exploration was performed, but an anterior gastrojejunostomy was performed. The patient's postoperative course following this was uneventful, and on her thirteenth postoperative day following the gastroenterostomy, she was dismissed from the hospital on a low fat, high protein, high carbohydrate diet, taking 50 mg. of Banthine 4 times daily.

Examination of the patient 2 months postoperatively revealed that the patient was gaining weight steadily and had no further pain. Her stools were somewhat fatty in description. Three months postoperatively, cholangiogram revealed normal common duct, and bile entered the duodenum in a normal manner. Upper gastrointestinal examination, 3 months postoperatively, revealed complete obstruction in the region of the pylorus with a wide duodenal loop, and the anterior gastroenterostomy was functioning well. Six months postoperatively, the patient had continued to gain weight and was irrigating her T tube daily. Cholangiogram was repeated at that time and revealed dye entering the duodenum promptly without any evidence of obstruction. One year postoperatively, the T tube was removed, and the patient was observed carefully without any unpleasant sequelae. The patient was followed carefully, following removal of the T tube, and examination 21 months postoperatively (9 months after removal of T tube) revealed that the patient was having no difficulty, was continuing to hold her weight, and had no recurrence of pain or vomiting. She has remained on a low fat diet and is continuing to take Banthine 4 times daily.

DISCUSSION

The occurrence of necrosis and perforation of the pancreatic portion of the common bile duct as a sequela of acute hemorrhagic pancreatitis immediately raises the question of possible etiology. Local infection with secondary necrosis and sloughing of the posterior wall of the common bile duct could conceivably occur, but it is surprising that it would occur so rapidly and in the absence of a generalized peritonitis. The possibility that the blood supply of the distal portion of the bile duct had been destroyed with the hemorrhagic necrosis of the head of the pancreas must be considered, but it is an unusual phenomenon to develop an infarction and necrosis of a portion of the common duct. Finally, one must consider the possibility of a local necrotizing agent. It is well known that erosion into major blood vessels is an occasional complication of hemorrhagic

pancreatitis, and it is probable that the erosion of a portion of a wall of the vessel is due to some local necrotizing agent—such as activated trypsinogen.

The methods of treatment of this complication are, of necessity, surgical. The finding of bile peritonitis with necrosis of the distal portion of the common duct by the surgeon at time of exploration must be dealt with according to the local condition. It must be remembered that when treating an extremely ill patient and when working with tissues which are markedly edematous and inflamed, the procedure which is performed must be a compromise of basic surgical principles with practical expediency.

1. Ligation of the common duct proximal to the defect and by-passing the biliary system probably with cholecystojejunostomy must be strongly considered. This possibility was entertained in the present case, but was abandoned due to fear for the viability of the gallbladder and its permanent usefulness as an intact structure. It was feared that eventual stenosis and stricture would occur within the gallbladder or cystic duct, so that cholecystojejunostomy could only serve as a temporary by-pass.

2. Cholecystostomy with or without ligation of the common duct is certainly a procedure which should be strongly considered and would have been a less radical procedure in the above case. It may be performed as a life-saving procedure, to divert the biliary stream from the region of the bile leak, and could be performed with the minimum of surgical intervention. In the above case, the common bile duct had already been opened approximately 3 cm. proximal to the region of the bile duct perforation, and, accordingly, ligation would have had to have been performed at the level of the cystic duct. Under these circumstances, only a short stump of common hepatic duct would remain for subsequent bile reconstruction.

3. Simple drainage of the area must be strongly considered and probably would be the simplest method of treatment for the patient in profound shock, and in whom the common duct had not already been opened.

4. Choledochoenterostomy, preferably choledochoduodenostomy, in the majority of patients, would not be practical due to the intense inflammatory reaction within the tissues, and the common duct is not so much dilated as edematous and enlarged, so that end to side anastomosis of the common bile duct and duodenum would be fraught with great danger.

The postoperative course of this patient was marked by the development of three major complications of acute hemorrhagic pancreatitis, namely; pseudocyst of the head of the pancreas with pyloric obstruction, secondary hemorrhage from necrosis of blood vessels, and toxic psychosis. These entities have all been described and fortunately the present case was successfully handled without any untoward difficulty.

One may question the advisability of prolonged T tube biliary drainage. The authors believe that in the situation which existed, biliary drainage was highly desirable for several reasons. The T tube draining the common duct and the region of the necrosis of the head of the pancreas allowed for more adequate drainage so that if an abscess formed in the lesser space, it would probably drain

into the common duct and into the T tube. T tube drainage also allowed for diversion of bile from the region of the pancreatic duct and from the region of the head of the pancreas so that no further bile leaked into the peritoneal cavity but was diverted to the outside. The presence of a T tube in such an instance acts as a splint, and it is quite possible that this may be an extremely valuable function of a T tube in the presence of a severely damaged structure, such as we had in this case. Finally, biliary drainage allowed for cholangiogram study of the biliary tree so that further complications could be anticipated and promptly dealt with.

SUMMARY

In summary, an unusual case of acute hemorrhagic pancreatic necrosis with necrosis of a portion of the head of the pancreas and necrosis and perforation of the common bile duct is presented. The patient's case was complicated by the development of a pseudocyst with pyloric obstruction, the development of toxic psychosis, and the development of secondary hemorrhage. The patient has subsequently been followed for almost 2 years, and, at the present time, is symptom free with no further evidence of residual disease.

Dept. of Surgery
Baylor Univ. College of Medicine
Houston, Texas

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GASTROJEJUNOCOLIC FISTULA: ONE STAGE RESECTION AND REPAIR

JOHN M. KESHISHIAN, M.D., OWEN GWATHMEY, M.D.

Washington, D. C.

One of the most serious complications of the ulcer diathesis is the formation of a gastrojejunocolic fistula. Because of the severe nature of this complication, and the grave prognosis if untreated, the diagnosis should be made immediately and surgical repair performed as soon as possible.

In the past, posterior gastroenterostomy was frequently used as definitive treatment for peptic ulcer. It was feared that many patients might ultimately develop gastrojejunocolic fistula if their ulcer diathesis persisted. This has not proved to be the case. Moreover, with the gradual abandonment of that procedure as definitive treatment for peptic ulcer fewer cases are expected.

The most common cause of this abnormal fistulous formation is the persistence of the ulcer diathesis. It may occur despite surgical treatment of the ulcer and, as such, it must be considered as a late complication of surgery.

Peristent high gastric acidity after gastroenterostomy or gastric resection and gastroenterostomy often leads to formation of a marginal ulcer. If this remains unrecognized and untreated, perforation of the ulcer into the colon may establish a gastrojejunocolic fistula.

The first gastrojejunocolic fistulas were reported by Czerny[5] in 1893. These were complications of the first gastroenterostomies performed by Wolfler in 1891 (posterior) and Von Hacker in 1885 (anterior).

The high incidence of marginal ulcer following posterior gastroenterostomy is well known. The progressive penetration of the ulcer and the anatomic rearrangement provides the ideal situation for the formation of a gastrojejunocolic fistula. According to Ransom,[4] marginal ulcers are also known to occur with duodenal exclusion procedures. However, he has observed no instances of gastrojejunocolic fistula following these procedures.

Marginal ulcers, following gastroenterostomy alone, will lead to gastrojejunocolic fistula in from 11 per cent to 15 per cent of the cases. The incidence of this occurrence is less, however, following gastric resection and gastroenterostomy. The inference is that adequate resection of gastric tissue decreases the acid formation.

Clinical Syndrome: The diagnosis of the abnormal fistulous connection can usually be made from the history alone. If there is a history of gastric disorders of long standing, in a male patient, who has had previous gastric surgery, suspicions should be aroused.

Further questioning will elicit a history of intermittent periods of distress and pain and, just prior to admission, relief of the intense, boring pain which usually signifies perforation.

From the Department of Surgery, The George Washington University School of Medicine, Washington, D. C.

The fistula may develop without warning, but once formed, the signs and symptoms make the diagnosis evident.

Diarrhea, nausea, belching, a feeling of being bloated, stercoraceous breath, the occasional passage of firm, hard stools and epigastric pain are common manifestations of the lesion. Sudden, progressive weight loss with resultant nutritional deficiency will occur.

Diarrhea is the most debilitating symptom and may prostrate the patient. It usually follows a meal and has a sudden onset. It is thought to be due to reflux of irritating colic contents into the jejunum with a reflex rush of peristalsis. Diarrhea is also a reflex from irritation of the marginal ulcer by ingestion of food. The occasional, firm, formed stool results from the *shunted* feces into the small bowel.

Pain is severest in the period preceding actual fistula formation. It is the characteristic sharp, boring epigastric, ulcer type pain. After perforation, the pain remains only as local tenderness, usually overlying the fistula.

Stercoraceous breath and belching result from the colic material entering the stomach. This usually precedes the diarrhea. The patient and persons in the immediate vicinity become unhappily aware of the odor.

Nutritional deficiency is the result of the negative nitrogen balance and secondary to inadequate food intake. Avitaminosis, low proteins and blood dyscrasias occur.

Roentgenologic Studies: The history should establish a correct diagnosis. In addition a barium enema study should be made; this will usually demonstrate the fistula. The barium swallow is not as satisfactory a method to demonstrate the gastric fistula, but occasionally if the stoma is of sufficient size, the tract can be demonstrated in this manner.

Preoperative Treatment: In every instance, once the diagnosis has been made, surgical correction is mandatory. Accordingly, preparations must be made for a definitive operation. First consideration should be given to restoration of the blood volume to normal levels. The serum proteins must be raised by the use of whole blood, serum albumin and plasma, massive doses of the soluble parenteral vitamins and vitamin K.

The value of adequate preoperative preparation was demonstrated by Gray and Sharpe,[1] with reduction of operative mortality rate from 61.5 per cent to 27.7 per cent. Bowel preparation should include the oral sulfonamides and wide spectrum antibiotics, as well as parenteral antibiotics. Sterilization of the bowel further aids in reducing the inflammatory reaction at the site of the gastrojejunocolic fistula. Oral sulfasuxidine, in addition, will keep the fecal material in the small bowel soft. Very often this *shunted* fecal material becomes quite hard, especially after the barium studies, and may be difficult to evacuate preoperatively.

Because of the high morbidity and mortality rates which previously attended this operation, Pfeiffer and Kent[3] suggested proximal colostomy as a preliminary step. It is believed that this would prevent reflux of colic contents into the small bowel, thus abating the diarrhea. Others advocate ileo-descending colostomy

as a preliminary step, thus diverting the fecal stream and reducing the irritation. All preliminary procedures have for their goal the possible added safety of step by step attack of the problem. There may be some justification for this in the severely debilitated patient who cannot tolerate long periods of surgery.

At the present time, however, when the patient has been properly prepared and made as ready as possible for operation, the single stage resection and anastomosis is much the preferred procedure.[2, 6] The same technical trends in the treatment of this disease apply in the single stage resection and anastomosis in the case of diverticula of the sigmoid colon. Previously, preliminary colostomy and long periods of drainage were the rule.

Operation: The gastrojejunocolic fistula exists either with a gastrectomy plus gastroenterostomy, or with gastroenterostomy alone. In the latter case, it is almost always a posterior gastroenterostomy. Unless there are technical contraindications, an adequate gastrectomy and gastroenterostomy should be performed. Any detachment of gastroenterostomy and restoration of continuity of bowel is to be condemned vigorously, since this resumes the old cycle. The afferent and efferent loops of colon and jejunum can be resected and included en bloc with the resected portion of the stomach. Primary anastomosis of these ends of bowel and gastroenterostomy is decidedly easier than tediously dissecting through scarred bowel wall. When gastrectomy has been performed previously, one must assume that it was inadequate and additional stomach should be taken.

In one of the cases reported here, an unusual situation existed. The patient developed a gastrojejunocolic fistula and moderately advanced bilateral pulmonary tuberculosis at approximately the same time. In view of the patient's double jeopardy, it was decided to perform surgical repair before the tuberculosis had healed. Since it is well known that adequate nutrition and long periods of rest can cure such infections and have done so in the past in many sanatoriums, the primary concern was repair of the fistula. As a forethought, it was hoped that a Billroth I repair after adequate resection would be feasible, since this would permit better absorption of food by restoring the normal gradient of the bowel.

This gradient has been shown to be one of the factors altered when analyzing causes of the "*dumping syndrome*" and incomplete digestion of foods.

CASE REPORTS

Case 1. A. K., a 45 year old white man, was admitted to the George Washington University Hospital on July 21, 1950, complaining of diarrhea of 4 months' duration. Twenty years prior to admission, the patient had a gastroenterostomy for persistent epigastric pain. There was temporary alleviation, but pain recurred intermittently. In 1947, a "*portion*" of his stomach was removed at another hospital, with improvement for several months. However, there was recurrence of epigastric pain which could be relieved by milk, cream and amphojel. Four months prior to this admission, the patient developed diarrhea, with 6 to 8 watery stools daily. Occasionally, there was passage of hard, formed stools. There was frequent nausea and belching followed by vomiting of fecal material. His weight dropped rapidly from a maximum of 199 pounds to 130 pounds. Pitting edema developed in both lower extremities.

On physical examination, the patient was well developed, but emaciated. The skin was loose with poor turgor and evidence of recent weight loss. There was an area of localized

tenderness in the epigastrium just beneath the old scars. The bowel sounds were hyperactive. Marked pitting edema of both lower extremities was detected, but there was no sacral edema. There was moderate edema of the forearms.

Laboratory data on admission revealed total proteins 4.8 Gm. per cent with albumin of 2.5 Gm., globulin, 2.3 Gm., hemoglobin, 96 per cent. Urinalysis was reported as normal. Gastric analyses revealed free acid 21°; total acid 27°. On July 31, after vigorous treatment, total proteins had risen to 6.0, albumin 3.7, and globulin 2.2. The patient had reached optimum state for operation and on Aug. 1, 1950, a single stage resection of the fistula, with involved portions of the jejunum, colon and stomach, was performed. An anterior Hoffmeister type of gastroenterostomy was used in the repair. The postoperative course was uneventful. There was no immediate recurrence of symptoms.

Case 2. M. S., a 55 year old white man, was admitted to the Glenn Dale Hospital on March 21, 1955, with a diagnosis of active tuberculosis. This patient had been transferred from the city hospital, after the diagnosis of tuberculosis had been made. Briefly, the history revealed that in 1929, he had suffered severe pain in the abdomen and a diagnosis of ruptured duodenal ulcer was made in a local hospital emergency room. He was operated upon and simple closure of the ulcer was performed. He was placed on a diet to which he adhered for more than a year, with weight gain and relief of symptoms. One year later, he again developed signs and symptoms of an acute ulcer and was admitted to the same hospi-

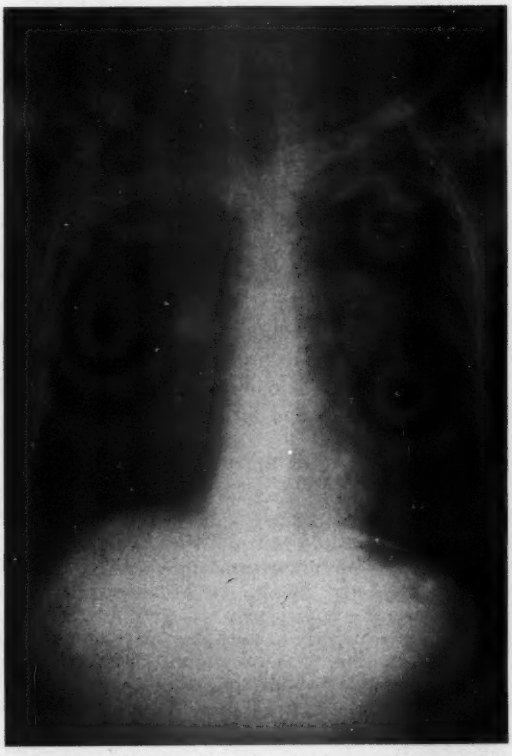

FIG. 1. Roentgenogram of the chest in Case 2, showing extensive bilateral, upper lobe tuberculosis.

tal. At that time posterior gastroenterostomy was done. He was again placed on a diet, but this time did not adhere to it. During the interim from 1930 to the date of admission, he had various jobs, wandering from one to another. He was a newspaper vendor at the time of admission. Moreover, he was an unreliable alcoholic addict with poor eating habits and long periods of drunkenness. He stated that much of his drinking was brought on by the pain in his abdomen. Approximately 2 to 3 years following the gastroenterostomy, his ulcer symptoms again returned. However, because of the poor result which he had had from two previous surgical operations, he thought that surgical treatment was not the answer to his problem. He had seen several physicians and been placed on numerous diets, but had abandoned all of them. Immediately preceding his admission to the Glenn Dale Hospital, he developed severe abdominal pain and diarrhea. On close questioning, he indicated that the diarrhea was the first symptom of his present illness. This diarrhea became so debilitating that he was confined to his bed and meals were brought to him, occasionally, by a friend. His condition became worse and he was sent to the city hospital by a visiting physician. Roentgenograms of the chest revealed bilateral, advanced tuberculosis (fig. 1).

Physical examination at the Glenn Dale Hospital revealed a frightened, scrawny, emaciated, hostile white man, who was acutely ill. The tissue turgor was poor and there was evidence of recent weight loss. His breath had a stercoracious odor. The abdomen was protuberant and the ankles were swollen with pitting edema (fig. 2). Immediate correction

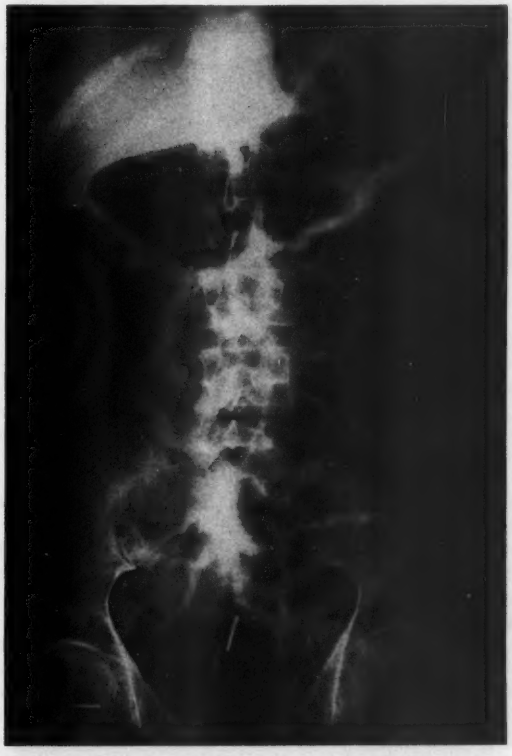

FIG. 2. Upright film of abdomen on admission in Case 2. Note extensive distention of stomach and colon. Small bowel pattern shows feces within lumen (not reproduced well).

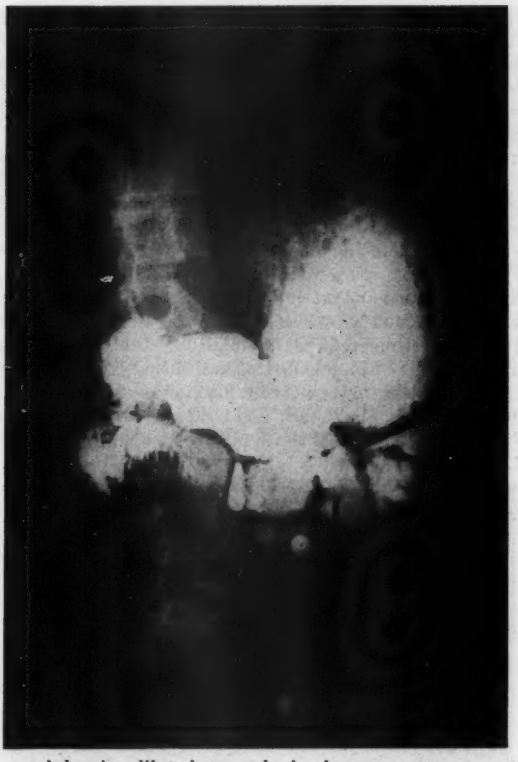

FIG. 3. Barium *meal* showing dilated stomach, duodenum, gastroenterostomy and colon

of the electrolyte imbalance was undertaken. A roentgenogram of the chest confirmed the advanced apical tuberculosis. Surgical consultation was requested because of the persistent nausea, vomiting and diarrhea. Barium swallow and gastrointestinal studies were done which confirmed a gastrojejunocolic fistula (figs. 3 and 4). Admission laboratory studies indicated anemia, dehydration and hypoproteinemia. The bromsulfalein was 4 per cent in 45 minutes. The total proteins were 5.5 Gm. per cent.

In addition to the abdominal disease, the tuberculosis was carefully evaluated. Isonicotinic acid hydrazide and streptomycin were given. Serial films of his chest were taken. It was apparent that the greatest danger to life was the gastrojejunocolic fistula, accordingly, preparations were made to repair this fistula. He was given whole blood and salt poor human serum albumin. Electrolyte studies were carried out daily until balance could be obtained. He was given massive doses of soluble vitamins parenterally; vitamin K1 oxide was given as well as Hesperedin and Rutin. While in the hospital, he developed massive areas of subcutaneous extravasation of blood. It was believed that this was due to the severe anemia which existed subsequent to the development of the fistula. These ecchymoses spontaneously disappeared with further doses of vitamin K and massive doses of vitamin C. This problem was of some concern to the surgeons since wound healing might be compromised if hematomata were in both bowel wall and the skin. In addition, the patients was placed on oral as well as parenteral antibiotics, beginning the day of the surgical decision. At the point of optimal preparation surgical intervention was undertaken. It is estimated that of the 96 days between admission and operation, 90 days were spent in preparation of the patient.

FIG. 4. Barium enema again demonstrating gastrojejunocolic fistula

FIG. 5. Postoperative film showing Billroth I repair

On June 25, 1955, through a right rectus incision, the abdomen was explored. The previous posterior gastroenterostomy was identified; the reaction about the stoma was so minimal that the defect could be palpated easily. Thumb and forefinger could easily roll around in the fistula, as one does when testing an anastomosis for patency. With this fortuitous finding, it was decided to perform a single stage resection of the involved loops, gastric resection and primary anastomosis. Since the stomach was tremendously flabby and atonic, it was thought that too radical resection might result in very little stomach, after the stomach had returned to normal size. In addition, it was decided to make the stoma twice the normal size for the same reason. Gastric resection was followed by a Billroth I repair, without difficulty (fig. 5). A Levine tube was passed through the anastomosis between the stomach and duodenum for feeding purposes and the patient was returned to the ward. The postoperative course was uneventful. Oral feedings were begun on the third day; the subsequent feedings coincided with a 7 day Sippy diet. There was no return of symptoms in this short interval.

Followup gastrointestinal studies showed a well functioning stoma.

COMMENT

We have been unable to locate the first patient since his discharge from the Out Patient Clinic.

The second patient had gained 50 pounds at the time of preparation of this manuscript (January 1956) and his intestinal disorders have abated. He is still receiving antituberculosis therapy.

SUMMARY

Two cases of single stage resection of gastrojejunocolic fistula are presented. Their signs, symptoms, treatment and management are presented. It is believed that single stage procedures are the treatment of choice, except in unusual cases.

Department of Surgery
George Washington Univ. School of Medicine
Washington, D. C.

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SPONTANEOUS PNEUMOPERITONEUM

JOHN H. SCHNEEWIND, M.D.

Chicago, Ill.

Spontaneous or idiopathic pneumoperitoneum is a syndrome consisting of free gas in the peritoneal cavity without demonstrable source. Clinically, there may be variable amounts of abdominal pain, tenderness and distention. However, on most occasions the abdominal findings are less pronounced than they are in peritonitis caused by perforation of a viscus. At operation, no cause for the free air can be found and frequently no intra-abdominal pathology of any kind is noted.

Leys[5], in 1944, reported 3 cases. The patient in case 1 had roentgenologic evidence of a deformed duodenal cap as well as free gas. This patient was not operated upon. The patient in case 2 showed evidence of an old duodenal ulcer when subsequently subjected to gastroenterostomy. The patient in case 3 entered the hospital with abdominal pain and distention of 3 days duration. At operation, gas rushed out of the peritoneal cavity; no lesion was found and a cecostomy was performed. In 48 hours recurrent pain and distention required another celiotomy. Again gas rushed out of the abdomen. The following day persistent pain and distention were treated by needle aspiration. The patient subsequently recovered. Barium study of the upper gastrointestinal tract 6 weeks later revealed no abnormality or free air.

Ayers and associates[1] reviewed the literature and reported a case in 1950. The patient was a 53 year old Negro man with a long history consistent with duodenal ulcer. Roentgenologic studies revealed a large amount of air under both leaves of the diaphragm, a deformed duodenal bulb and an enlarged stomach. The patient refused operation and was kept in the hospital for 3 months. During this time the air in the peritoneal cavity remained constant. A month later a plain film of the abdomen showed no evidence of pneumoperitoneum. No evidence of gas cysts of the intestines was found in this patient, but the authors suggest that cystic disease of the intestinal tract might be found in some patients as a cause of the spontaneous pneumoperitoneum.

Maddock, Bell and Tremaine[6] have made a thorough study of the source of gastrointestinal gas. They state that excessive amounts of air may enter the stomach by frequent swallowing and also by attempts to breathe against a closed glottis while the superior esophageal sphincter is relaxed. The authors reported a case of massive pneumoperitoneum due to spontaneous rupture of a tremendously dilated stomach which occurred during a minor operation under sodium pentothal anesthesia. They believed that the cause of the gastric dilatation was relaxation of the superior esophageal sphincter and respiratory aspiration of air.

CASE REPORT

Case 1. A 25 year old Negro woman, who previously had been an obstetrical patient at the Research and Educational Hospitals, was seen in the Emergency Room complaining of

From the Department of Surgery, University of Illinois College of Medicine, Chicago, Illinois.

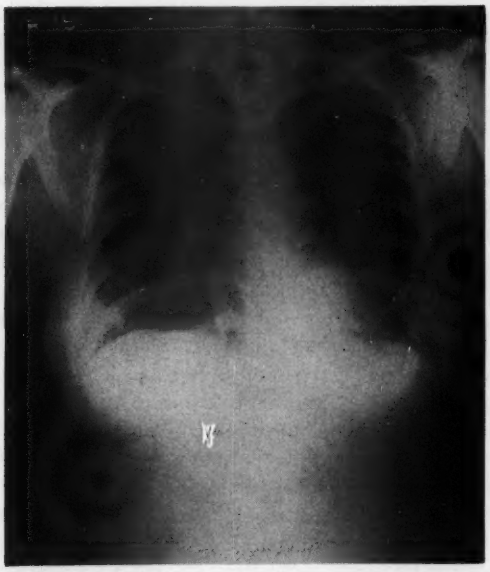

FIG. 1. Patient S. D. Upright film of chest and upper abdomen showing free air under right diaphragm.

lower abdominal pain. Except for poor appetite during the past 3 days, she had been well until the morning of admission when she suddenly developed sharp and severe left lower quadrant pain. The pain had persisted 10 to 11 hours prior to admission and had been continuous and not cramping. There was no radiation of the pain. About ½ hour following the onset of pain, she vomited a green, bitter-tasting material. There had been no bowel movements during the day of admission. There were no urinary complaints. Past history revealed that about 1 year ago she experienced a similar episode which subsided spontaneously.

Physical examination: Physical examination revealed a pulse of 100, blood pressure of 132/85 and respirations of 25. Temperature on admission was 100 F. Heart and lungs were not remarkable except for the tachycardia. The abdomen was moderately obese, somewhat distended, and diffusely tender in both lower quadrants, especially on the left. There was some guarding but no rigidity. There was rebound tenderness on the left but no point tenderness. Bowel sounds were normal. Pelvic and rectal examinations revealed bilateral adnexal tenderness most marked on the left. There were no palpable masses. Roentgenogram of the abdomen revealed free air under the right diaphragm (fig. 1). At operation no perforation in the gastrointestinal tract was found. The entire abdomen was essentially normal except for a moderate mesenteric adenopathy. No diverticula were identified.

The patient had an uneventful postoperative course. Ten days after operation barium studies of the entire gastrointestinal tract were interpreted as normal.

DISCUSSION

Several possible sources of the gas in cases of this kind have been suggested. They include 1) escape of gas from minute perforations of the stomach or upper small intestine, 2) from the chest via a diaphragmatic hiatus and due perhaps to rupture of an emphysematous bleb, and 3) vaginal through the fallopian tubes

following douching. It is conceivable also that vigorous air-swallowing might be a cause in some cases. We recently encountered a patient in whom a nasal oxygen tube was erroneously placed in the hypopharynx. Severe distention and marked pneumoperitoneum resulted. However, at operation no perforation of stomach or intestine was found. The patient recovered without further incident. We do not consider this a case of spontaneous pneumoperitoneum because, while no perforated viscus was found, the source of the gas was obvious.

It appears that when coincidental pathology is present, it is most frequently related to old duodenal ulcers with varying amounts of fibrosis. This suggests that in some cases of spontaneous pneumoperitoneum partial duodenal block may be a factor.

If a patient presents pneumoperitoneum with no obvious cause, such as a perforated viscus or penetrating injury to the abdomen, it might be possible and advisable to delay or avoid celiotomy, providing the patient is kept under continual careful observation. Signs of peritoneal irritation, fever, tachycardia or abdominal pain would probably require immediate exploration.

SUMMARY

A case of spontaneous pneumoperitoneum is reported. At operation no cause for the free air was found and the patient recovered without complications. Subsequent barium study revealed a normal gastrointestinal tract.

Univ. of Illinois College of Medicine
Chicago 12, Ill.

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ACCIDENTAL HEAD INJURIES IN OCCUPANTS OF AUTOMOBILES

A REPORT OF TWO-HUNDRED AND SEVENTY-THREE CASES

JACOB KULOWSKI, M.D.

St. Joseph, Mo.

Failure to distinguish between facial[24] and actual head injuries has probably resulted in erroneous reports with regard to the latter's incidence in series of cases. Actually, about 50 per cent of these cases involve the facial areas (fig. 1) and not the head proper. Moreover, in regard to traffic injuries, both pedestrian and motorist casualties have been lumped together despite the fact that the forces involved in the production of these respective subgroups differ from one another.[39] Finally, failure to stress the variable morbidity of these injuries has sometimes resulted in considerable confusion in regard to external, skeletal and intracranial injuries and/or complications.

Relevant data in this paper are based upon 273 motorist casualties with true head injuries in a total series of 661 persons, or 41 per cent of the latter (approximately the same number also received facial injuries).* The sexes were about evenly divided, and all decades through the ninth were represented, with the greatest frequency in the second, third and fourth decades.

ETIOLOGY

Table I attests to the universality of forces[20] which produce head injuries in occupants of ground crash vehicles despite the variability of the principal impacts encountered. In other words, it seems to make little difference from the standpoint of the morbidity pattern what the principal impact was that caused it.† The implications of this statement are far reaching. In the first place, this means that a definite pattern of head injury exists in motorist casualties on a statistical basis. Second, clinicians should be alert to possible head injuries in all motorist casualties regardless of the mechanism of accident. Third, the more or less fixed pattern of injury strongly suggests that head injury is likely to be a function of automotive design and the occupant's relationship to the various interior impact areas.[2, 37, 3, 4, 5] Fourth, if this is true, then, modifications of these areas should have a moderating effect upon the resultant head injuries[21, 3]. Fifth, this clinical trend ought to guide and/or supplement experimental investigations along these lines. It does not appear to be necessary to try and reproduce all of the mechanical variables which are involved in field crashes. The

* People injured in ground automotive vehicles may be divided into four groups: first, those who escape injury; second, those who die instantly or at the scene of accident; third, those who die later on; and, fourth, those who recover after injury. In a clinical sense, then, the term survivors refers only to the last subgroup. The present discussion concerns only survivors (661 people) who had been injured severely enough to have been admitted to the Missouri Methodist Hospital from late in 1949 through 1954; and, had been treated by various members (including the author) of the medical, surgical and dental staffs. Data were extracted from the medical records of these people, and from 29 autopsy records.

† Results are tabulated as percentages based upon the number of injuries, not persons.

TABLE I

Nature of head injury as related to principle impacts under crash and/or up-set conditions

Type of Collision or Up-set	Topical Injury*	Skeletal Injury	Shock	Cerebral Concussion	Internal Injury
	%	%	%	%	%
Forward vehicular collisions	54	8	5	30†	3
Rear-end collisions	56	11	0	22	11
Vehicular collision from side	58	0	0	26	16
Collision and roll-over	63	0	0	37	0
Collision with ejection of occupant	48	3	0	42	6
Forced off the road, with and without collision	40	0	10	50	0

* Includes laceration, contusion and abrasion of the scalp or forehead.

† Two postconcussion psychoses from this group.

Note: Mechanical failures as causes of these accidents were negligible. Notable also was the fact that the vast majority of the accidents involved two or more drivers.

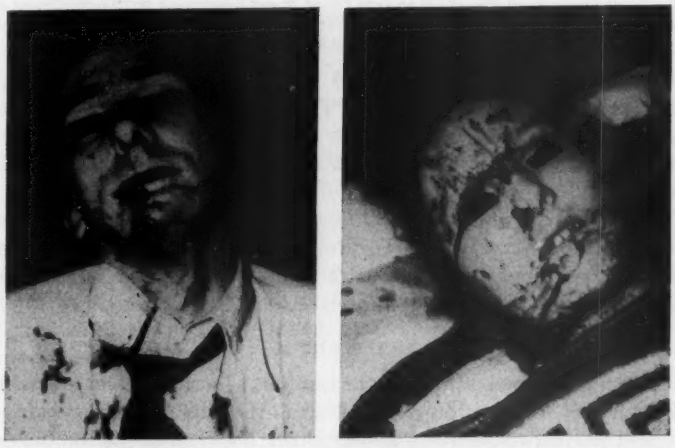

FIG. 1. Fairly typical appearances of front seat motorist injuries: A. driver, with compound fracture of the mandible from impacting the steering post; B. front seat guest passenger (wife) with multiple facial lacerations from the windshield, shock, fracture of the clavicle and ankle. Facial and head injuries present a Janus-like problem for diagnosis, treatment and prophylaxis.

simplified and controlled situations employed by research workers who are studying head injuries of mechanical origin are fundamentally sound[21, 35].

Among the physical factors which predispose occupants of crash vehicles to injury, direction of force, area of the body to which it is applied and to a lesser extent the surface area affected (psi) probably are largely determined by seating or the relationship of people to the various impact areas which limit the compartment (injury potentials of interior automotive design)[37, 38, 4]. In this series of head injuries the seating of the various occupants was as follows: drivers, 38

FIG. 2. Basal fracture of the skull in front seat guest passenger with self decompressing complicating hemorrhage from the left ear. Principal impacts in figures 1 and 2 were forward types due to high-speed collisions with other vehicles.

per cent; right front seat passengers, 34 per cent; back seat passengers, 8 per cent; and, unclassified ones, 21 per cent. The preponderance of front seat casualties (fig. 2) would seem to implicate the chief injury producing forward structures of modern automotive ground vehicles; that is, windshield, dash and steering controls.

It is also interesting to note that 75 per cent of the drivers were males, and approximately 75 per cent of the right front seat passengers were females. This was also true, regarding the latter, with respect to back seat passengers. The implications in regard to driver proficiency should be quite evident.[37]

EXPERIMENTAL

As has been mentioned injuries of the head may be classified as external (topical), skeletal and internal. Since the skull is the natural barrier between the external and internal structures, major attention has been focused on the mechanical factors which result in linear fractures here. The work of Gurdjian and his associates has established five fundamental relationships between force (energy input) and skull fracture[11, 12, 13, 14, 15, 16]: (1) the overlying soft parts are efficient absorbers of impact energy, (2) linear fractures are due to excessive tensile stresses, (3) the practical elastic or tolerance limits of the skull as a whole is an energy input of approximately 400 inch pounds (weight times distance), (4) once linear fracture has occurred, it requires very little more energy to affect extensive damage or disruption, (5) the fracture site can be predicted from a knowledge of the impact areas. The mechanical aspects of skull fractures, as shown by Gurdjian and his associates, have been substantiated by the investigations of the Cornell Crash Injury Research group at Buffalo[1].

With regard to what takes place inside the skull, it has been known for some

time that concussive effects probably derive from sudden rises in intracranial pressure, because the incompressible intracranial contents have no avenue of escape when deformation-dissipation processes occur after external forces have been applied to the head. Denny-Brown pointed out some years ago that the rate of change of velocity (acceleration or deceleration), and not the momentum was the critical factor in the production of physical concussion[7]. According to him, acceleration of the head from 0 to 28 feet per second produces cerebral concussion. In his experimental animals, Denny-Brown was able to prevent both skull fracture and concussion by fashioning a small helmet for them from a tobacco tin. Scott,[34] a year before Denny-Brown, stated that concussion would result when the intracranial pressure rose above or to the systolic blood pressure. Currently, Gurdjian and his associates[17], are also engaged with this problem. They believe that impacts on the head which is fixed has longer acting forces on it than when the head is free to move; because, cranial restitution is facilitated in the latter condition.

PATHOLOGY*

Relevant data in this regard were extracted from the autopsy records of 29 motorist fatalities. The incidence of fractures and internal injuries were the most critical criteria in regard to the forces involved. The frequency rates of fracture in the various parts of the body were: chest, 69 per cent; extremity, 50 per cent; head, 32 per cent; pelvis, 32 per cent; and, face, 14 per cent respectively.

It should be remembered that brain injuries can occur without skull fractures and vice versa. In other words, fractures may be too severe a criterion of the results of automotive crash forces. In this series of fatalities, 7 persons or 25 per cent of the injuries to the brain were not associated with skull fractures (fig. 3). Several of the latter cases resembled so-called blast injuries. Two instances of brain injury without actual skull fracture deserve special mention. The first occurred in a child who had been sitting in the back seat when her car had crashed into the back end of a stalled truck. In the second case, another child had been ejected from the back seat (double jeopardy) and run over (with separation of the cranial suture lines, but no fractures). Thus, the back seat can kill (fig. 4) when occupants are catapulted over the front seat and/or forcibly ejected from the vehicle during impact or up-set.

More specifically, internal injuries in this series of deaths, numbered 19 intracranial, 26 above the diaphragm, 2 of the diaphragm, 25 below the diaphragm and 3 of the extremities. Approximately 75 per cent of these primary injuries occurred above and below the diaphragm. From the standpoint of primary injuries, head injuries tend to trail those of the chest and abdomen in rank. There were 18 injuries of the brain, 15 of which were contusive; and, 1 laceration of the optic nerve. Petechial hemorrhages (contusive?) were very commonly noted in the region of the pituitary body; and, probably resulted from convergence of

* Autopsy records were obtained from three sources: 12 from the University of Kansas Medical Center; 15 from the Missouri Methodist Hospital; and, 2 from the St. Joseph Hospital.

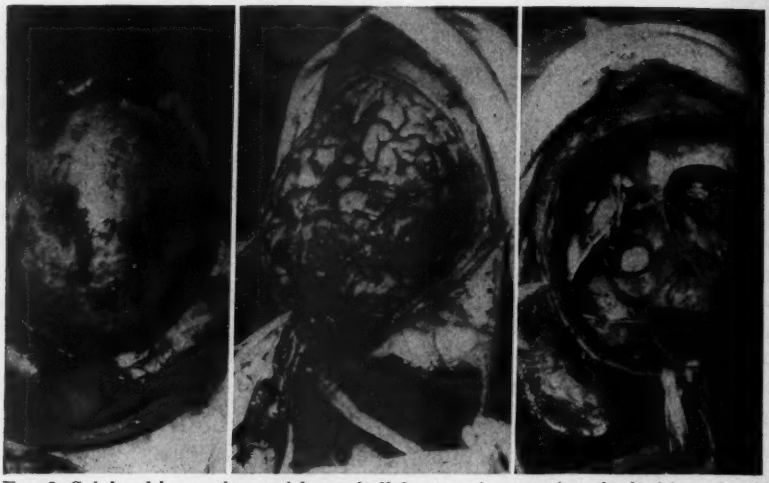

FIG. 3. Subdural hemorrhage without skull fracture in motorist who had been forcibly ejected from the vehicle; died 12 hours after admission to the hospital with multiple internal injuries both above and below the diaphragm.

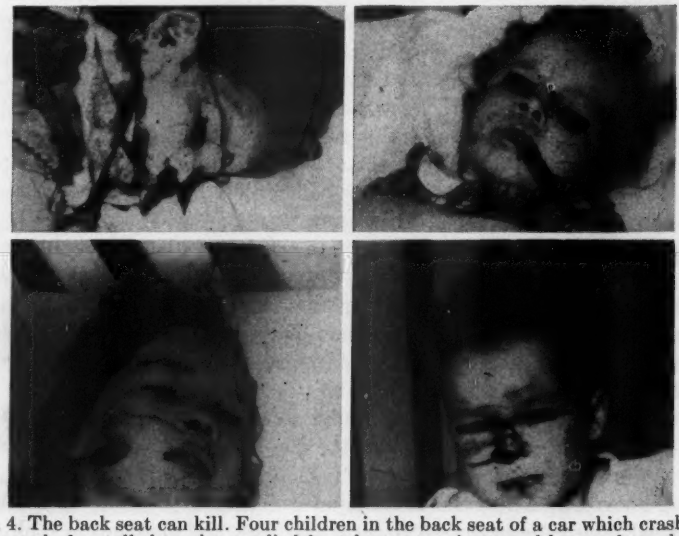

FIG. 4. The back seat can kill. Four children in the back seat of a car which crashed into the rear-end of a stalled truck; one died from hyperpyrexia several hours after admission. Autopsy showed only scattered petechial brain hemorrhages.

forces to this area due to the peculiar structural characteristics of the vault and base of the skull. Also, the frequency of contusive lesions would indicate that cerebral concussion must be commonly associated with such gross, but reversible, lesions; as well as more molecular types of pathology.

Complications, which must always be distinguished from primary injuries, comprised the following direct types: 27 hemorrhage; 3 cerebral edema; and, 3 cerebral softening; and the following indirect complications: 3 fat embolism; and, 2 meningoencephalitis. Attention is directed to one of many paradoxic results of motorist crash forces. From the standpoint of primary injury, the critical area of the body seems to be that of the chest; especially the mediastinal structures. On the other hand, considerations of secondary complications, regains for the head its traditional rank; because of the dire effects of space occupying bleeding within the cranial cavity. In this series, 10 of the hemorrhages were considered by me to have been space occupying; 9 subdural and 1 epidural. There were 5 subarachnoid and rather frequent petechial hemorrhages.

INCIDENCE OF AND TYPES OF LESIONS IN SURVIVORS

Table II establishes the frequency and types of head injuries in this series 273 motorist casualty survivors. These statistics are based upon the number of lesions and not the number of persons affected. The areas affected were the forehead, ears, scalp and skull (vault and base). Topical injuries refer to contusions, abrasions and lacerations, which would appear to be the common denominators in regard to head injuries. The relatively low incidence of skull fractures in survivors can be explained by the fact that facial injuries exert a buffer or sparing action in that regard; and, because of the high mortality rate attending complicated head injuries.

The high incidence of cerebral concussion re-emphasizes the increasingly better known fact that serious internal injuries may occur without concurrent local fracture or other local evidences of violence. In this series cerebral concussion was noted in about one third of the cases (fig. 5). In 43 per cent of these people there were no associated contusions, abrasions or lacerations of the head. Forty per cent did have lacerations; and, in 17 per cent there were concurrent contusions and abrasions.

TABLE II

Correlation between type of head injury and seating in 273 motorist casualty survivors

Seating of Occupants	Number	Topical Injury	Skull Fracture	Cerebral Concussion	Intracranial Injury	Total Number of Injuries
		%	%	%	%	
Drivers	104	53	5	40	2	173
Front seat passengers	94	63	7	26	4	142
Rear seat passengers	21	62	8	27	3	26
Unclassified	54	66	1	30	3	70
Total	273					411

Note: Of the 273 people with head injuries there were 244 topical injuries (59 per cent), 21 fractures of the skull (5 per cent), 134 with cerebral concussion (33 per cent), and 12 intracranial injuries (3 per cent). Of the 21 skull fractures, ⅓ involved the vault, 3 were basal and the remainder were unclassified.

FIG. 5. Severe cerebral concussion in front seat passenger who was involved in a forward type vehicular collision; also unusual abrasive contusion of the neck, shock and femoral fracture. Recovery without cerebral sequellae.

Shock[9] was reported in 21 persons (8 per cent); that is, in 8 drivers; 12 right front seat passengers, and 1 in the back seat. Only 12 per cent (32 persons) received injuries of the head alone. Eighty-eight per cent received other body injuries in addition to those of the head. Based on the number of injuries, concurrent injuries occurred as follows: extremity 34 per cent; face 28 per cent, chest 12 per cent, multiple topical lesions 11 per cent, trunk 5 per cent, neck 5 per cent, pelvis 3 per cent, other internal injuries 1 per cent and abdominal injuries less than 1 per cent. This parallels roughly the injury pattern observed in the entire series of survivors (661 cases) which made head injuries second only to those of the extremities and equaling the facial ones. Combined, about 80 per cent of the series as a whole received facial and/or head injuries.

From the standpoint of seating, it should be noted that every area of the car is dangerous in regard to head injury. About 40 per cent of all the injuries were received by drivers; and, head fractures are a little more frequent among front seat passengers. The reverse is true in regard to facial lesions. However, viewed from the standpoint of degree of morbidity involving the entire series of survivors, seating does not determine how badly injured the occupants will be. It was found that 45 per cent had mild to moderate injuries; 45 per cent had moderate to severe injuries; and, 10 per cent received severe to dangerous injuries. The percentages of these three categories ran about the same regardless of the seating. In other words, seating did not determine the degree of morbidity. However, from the standpoint of mortality, drivers seemed to get the worst of it. It has been stated that survival under crash conditions is three times more likely in the back seat than in the front one.

It cannot be over emphasized that the mortality rate derives from the small

but lethal class of dangerously injured people. The overall mortality rate in the entire series of cases studied by me was 3.4 per cent; that for the seriously injured group was therefore 34 per cent. If we are to lower the mortality rate in motorist casualties, that is by treatment, it will have to be done in that category of casualties. The therapeutic needs can best be judged on the basis of the mortality rate differentials[27], and not upon survivors.

THERAPEUTIC IMPLICATIONS

On the basis of the already mentioned 29 fatalities, three mortality rate subgroups emerged; from the standpoint of time interval between injury and death; i.e., immediate, intermediate and delayed. Considerations of these categories have diagnostic and prophylactic implications as well as those of treatment. In this small series 3 persons (10 per cent) died immediately or at the scene of accident; 19 (66 per cent) died within 48 hours after injury; and, 7 succumbed (24 per cent) up to 8 days afterward.

The immediate fatalities impugn the ultimate failure between human tolerances and automotive design. In these 3 patients, mediastinal and other internal injuries—not head injuries—were the respective causes of death. The crashes were all highspeed forward impacts in which the steering controls had been severely engaged and impacted. Deaths were due to primary shock[30, 31]; i.e., massive and irreversible (?) circulatory failure in the fullest sense of the term (primary shock). The only hope for this class of casualty is prophylaxis along two intersecting lines of thought and action; that is prevention of accidents in the first place (primary safety), and reduction of injuries under crash conditions (human and crash-impact engineering or supplementary safety). In capsular form this would include reasonable speeds for driving, driver proficiency (education and training), adequate medical standards of licensure, and crash-proofing of vehicles (revisions of external and internal automotive design and structure, safety barriers between occupants and impact areas (safety belts etc))[3, 6, 14, 21, 26, 36].

Intermediate deaths, on the other hand, focus attention upon first aid[28, 33] and emergency care; and, delayed fatalities point to definitive treatment. The full impact of this statement rests on the fact that approximately 85 per cent of motorist fatalities occur after—not during—the accident. Primary shock was still the obvious cause of death in 10 persons who died within several hours of their respective hospital admissions. Their lesions were 4 intracranial, 7 intrathoracic, 3 abdominal and were associated with 5 massive hemorrhages. In the intermediate mortality rate for those who lived longer (9 cases), the intracranial lesions predominated. However, already, conditions which are operative in secondary shock had made their appearance (circulatory failure, fat embolism, lower nephron nephrosis and pneumonia). The rapidity with which these factors appear is seldom fully appreciated by medical attendants.

First aid is virtually nonexistent in regard to motorist casualty survivors; despite the fact that so many of these persons receive obvious injuries of the extremities. The solution to this shocking situation (moral and physiologic) ought to come from the combined efforts of medical groups, Red Cross, Civil

Defense, hospital and nurses groups, and lay interests. It would be most advantageous to render some training in modern (simplified) first aid methods to those who are usually available at highway accidents, such as truck drivers, bus drivers and ambulance attendants. This could also be made part of 4 H activities for the younger people in the rural areas.

An integral part of first aid is transport to the hospital. Simple as this phase is, very little thought has been given to it. I refer, of course, to the observation that all patients except those with the most serious head injuries will tolerate transport in modern ambulances well[23]; if they are turned on their sides and the airway kept open. It is most important to get these people where they can get the benefit of neurosurgical diagnosis and treatment. For that matter, all seriously injured motorists should be taken to hospitals where personnel and facilities for adequate care are at hand; not later on, but from the scene of accident. Such proper initial action aids emergency care. Once the patient reaches the hospital, it is emphasized that the same humanitarian principles, on which first aid is based, be attained and/or maintained there.

Emergency care obligates hospitals to supply the opportunities for the immediate treatment of primary shock (plasma expanders and large amounts of blood must be available), cardiac arrest, pulmonary ventilation (tracheotomy as indicated); and, cerebral decompression. The latter should not be undertaken lightly; especially without neurosurgical consultation, because there is usually time for that and for proper preparation of the patient. Clinical and pathologic studies indicate that shock, cardiac arrest and pulmonary ventilation are the major conditions of urgency; and should be given immediate attention in the outpatient or admission department of the hospital[19]. At the same time, there is to be no let down in the care of simpler or run-of-the-mill survivable injuries.

Those who survived after 48 hours but died within 3 to 8 days after admission, in this series of fatalities, were younger individuals (only one gerontol). Conditions contributing to their deaths were intrathoracic 7, intracranial 4, abdominal 2 and massive hemorrhages 2. Conditions contributing to secondary shock included meningoencephalitis 2, fat embolism 3, pneumonia 3 and lower nephron nephrosis 3. Obviously, definitive diagnosis and treatment demands continued alertness and awareness of distinctions between primary and secondary conditions; which relentlessly encroach upon the patients' limits of factors of safety or survival quotient. Ultimate clinical awareness along these lines must depend upon valid pathologic data; which, to-day, is buried in a mass of unrelated recorded matter in the literature and hospital records. When this data is brought out, motorist injuries and complications will go to the forefront of clinical attraction, where these lesions rightly belong. In regard to the head injury itself, it is helpful to remember that most of these injuries are contusive, not lacerative. Nevertheless, space occupying hemorrhages are being overlooked; especially the subdural ones.

In this connection, Gurdjian and his associates[10] correlated the pathologic features of serious head injuries and complications with surgical treatment. Two observations were emphasized: (1) that there was a combination of pathologic

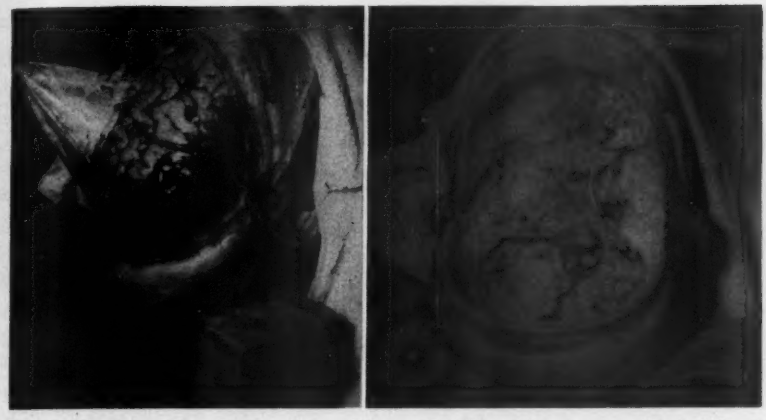

FIG. 6. Massive subdural hemorrhage with associated basal fractures of the skull in adult driver who was involved in a severe forward vehicular collision.

processes inside the head in practically every fatal case; and, (2) although the pathologic processes are variable, there is always gross evidence or evidences of it. These authors gave three rules as a guide to surgical care: (1) massive hemorrhage is usually on top of the brain and is therefore amenable to surgical treatment; (2) epidural hemorrhage should be explored for in the fracture line; and, (3) subdural hemorrhage, on the other hand, is often opposite to the gross fracture line (fig. 6).

An appreciation of the secondary complications tends to stress the value of teamwork in the treatment of any serious motorist injuries. The role of the neurosurgeon, in connection with head injuries should be obvious. That the internists should be made part of any team which undertakes to care for seriously injured people has not been emphasized enough. I concede that alertness for the more subtle encroachments by embolic, nephrotic, psychologic (or psychosomatic) and pulmonary (with or without mediastinal conditions) participations are in the domain of internal medicine rather than surgery; and, welcome their aid and supervision in these cases. As a matter of fact, the longer the patient survives (in the first week of his injury and post-traumatic period) the more likely is he to become primarily a medical rather than a surgical case; from the standpoint of survival. This, in turn, calls for a more aggressive diagnostic attitude in dangerously injured people.

Diagnostic measures ought never to be withheld because of real or supposed added risks entailed in doing so, because active and specific treatment of these people, especially, must depend upon accurate diagnosis, and an over-all clinical evaluation of the situation. The frequency of chest and mediastinal injuries (about 30 per cent of the latter in the small series of fatalities already mentioned[27]) begs for baseline roentgenographic and laboratory investigations (including EKG) from the very beginning of observation in the hospital. Both neurosurgeons and internists should have a major share in the care of those with serious head injuries.

PROPHYLAXIS

Since head injuries of mechanical origin have actually bridged the gap between engineers and medical groups, it seems appropriate to discuss prophylaxis further. It must be clear, however, that the chest—not the head—is the most critical area of the body from the standpoint of motorist injuries and complications. Nevertheless, intrathoracic rank has been achieved in part from the observation that in the final analysis death results, even from head injuries[18, 22, 32], from serious intervention with cardiorespiratory function. Clinical appreciation of the reciprocal relationships which exist between cerebral and cardiorespiratory functions (adequate pulmonary ventilation) cannot but help to reduce the mortality in dangerously injured people. Herein, then, are clearly defined the limits of factors of safety of the human body[8]. In other words, from the standpoint of mortality (as distinguished from morbidity) the chief efforts toward prophylaxis in automotive safety design should be concentrated upon the prevention and/or moderation of head and chest injuries. The simplest way to achieve this, from the standpoint of crash-proofing, is by adequate safety barriers between occupants and impact areas (including sufficient area between them). When this fundamental requirement has been accomplished, closer scrutiny should be made in regard to other impact injuries (less lethal and/or disabling) which make up the motorist injury patterns in order that a further reduction or amelioration of the morbidity may be made. A self perpetuating safety cycle will then have been established.

SUMMARY AND CONCLUSIONS

The present discussion has been based upon 273 motorist casualty survivors with head injuries (as distinguished from those of face) and 29 motorist fatalities. Head injuries are second to those of the extremities and equal those of the face in motorist survivors. The variability and relative moderation of injuries in survivors contrasts sharply with the dangerousness of the injuries and complications observed in fatal cases. In the former group, then, the term head injury should not be considered to be synonymous with skull fracture and/or brain injury; as is sometimes apt to be the case unless the morbidity of these lesions is clearly stated. The opposite obtains in regard to internal injuries in the fatal group. Careful considerations of the latter recall that causes of death, in order that they may be interpreted on rational clinical bases, are either those which contribute to primary shock or secondary shock. From this standpoint it becomes relatively easy to point out needs along lines of first aid, emergency care and definitive diagnosis and treatment. Recovery after crashing will depend to a great extent upon the quality and continuity of treatment from first aid to definitive treatment and ultimate rehabilitation.

This leaves two other intersecting phases of prophylaxis; that is prevention of accidents and reduction of injuries. Nothing can supersede primary accident prevention. The weakest link here is the human ingredient. Medicine's chief role should be the development of adequate medical standards of licensure.

Second, medical groups ought to be aware of what human-engineering[29] is trying to do; and, give what aid they can along this line.

The same may be said of so-called crash-impact engineering which aims to reduce crash injuries and deaths by delethalizing automotive design. In this regard also, medicine should endeavor to cooperate to the fullest extent with those who are trying to integrate medical and engineering data for the above purposes. The weakest link would seem to be a lack of valid medical, and especially pathologic, data in regard to motorist casualties. This can easily be remedied when writers on trauma of mechanical origin will separate these injuries in accordance with their respective causes.

Actually, medicine is expected to widen its clinical borders so to speak to include primary and supplementary methods of prophylaxis as has been so notably done by aviation and military medicine. It seems logical to include etiology and prophylaxis with diagnosis and treatment. The probability that improved automotive design will reduce the immediate mortality rate and increase the diagnostic and therapeutic challenges surpasses logic. The need to widen medical interests in motorist casualties is mandatory[23].

415 Corby Bldg.
St. Joseph, Mo.

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EDITORIAL

HOMO SKIN GRAFTS FROM POSTMORTEM SOURCES FOR SEVERE BURNS

At the present time a most important element in the use of homografts of skin for severely burned patients is to try to get the public to accept the idea of allowing superficial layers of skin in unexposed areas to be removed, after death, for such use. Also important, is for the profession, as a whole, to accept the idea of this surgical principle of utilizing split grafts from postmortem sources when homografts might be of use, or even life-saving, in dealing with severely burned patients.

Only recently there was a front page news item from another area of the country concerning the fact that 100 persons had volunteered to give their skin for a burned child. It is our belief that taking homografts of skin from live donors is no longer necessary and that, in instances of need, the homografts may be obtained from postmortem sources. This is possible because of a differential of time, between general death and final death of skin, of several hours, or even days, during which time the skin grafts can be removed. Such removal does not interfere with the patient, with the relatives, or with the funeral director, because only partial thickness split grafts are taken from unexposed areas.

These grafts have been used to save lives by offering *"biological dressings"* for wounds that otherwise might be fatal, and if large supplies of the postmortem grafts were available, they could be used as dressings on burns of lesser extent and depth.

It is a dignified surgical procedure of removal of the split skin grafts from the person who has died, and covering the wounds of the patient who might otherwise die. Human worth and dignity thus may be carried even beyond death, in that the patient who has just died, has saved the life of another patient even after his own death.

Naturally one thinks of storage of these grafts and this is desirable. They may be simply stored for 3 weeks at normal refrigerator temperature +4 C. and still used. Other methods of storage are being studied, and nonviable freeze-dry preparations may be resoftened with saline solution and used with almost as long advantage as viable grafts. At present we believe that the closer to the time of removal from the donor the graft is used, the longer it may last; but there is some evidence that retention of the skin, for a few days, on a refrigerated body may lend some days to the persistence of the grafts, when used.

It is not meant that this is the only part of, or the answer to, the care of severe deep burns. All the elements of care are still needed, but this offers a way of closing the wound temporarily without having to resort to multiple live donors with the attendant anesthetics, operations, and debilitations. It also offers the skin more readily and easily obtainable so that perhaps the advantage of homografts can be given to more patients.

There are many details to be worked out as to most advantageous times of obtaining and storage and utilizing these homografts from postmortem sources. But it is pointed out that the method is open to use to anyone or any hospital that will go to the trouble of instituting the idea, the procurement, and the utilization of these grafts, that may be life-saving. Any service that does this may be said to have a skin bank in operation, and further details will be worked out with experience. (It seems unnecessary to say that homografts last only 3 to 8 weeks, except in identical twins, and that the patient's own skin will have to be used for permanent healing.)

Our report is thought to be the first record with illustrations of successful and possibly life-saving use of postmortem grafts in the literature. Previous suggestions had ranged from bizarre to unnecessary experimentation on patients with small lesions where autografts could have been used and healing completed. There is no reason to subject patients or volunteers to experimentation for any data where small laboratory animals can be used, including the transplantation of human skin to the animals for observation of its properties. There is no reason to waste homografts when the patients' own skin could be used and permanent healing obtained.

With Dr. Minot P. Fryer and with the assistance of others working with us we have tried to report these observations.

We believe the use of homo skin grafts from postmortem sources may be taken as a proved surgical procedure to save lives in severe burns, and we believe the method can be utilized in specific needs by any surgical service now without delay, without special equipment, and without waiting for further details of preservation of the grafts.

JAMES BARRETT BROWN, M.D.

100 N. Euclid Ave.
Saint Louis 8, Mo.

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